FILE 'MEDLINE' ENTERED AT 07:44:19 ON 01 OCT 2004

FILE 'BIOSIS' ENTERED AT 07:44:19 ON 01 OCT 2004
Copyright (c) 2004 The Thomson Corporation.

FILE 'DGENE' ENTERED AT 07:44:19 ON 01 OCT 2004
COPYRIGHT (C) 2004 THE THOMSON CORPORATION

FILE 'EMBASE' ENTERED AT 07:44:19 ON 01 OCT 2004
COPYRIGHT (C) 2004 Elsevier Inc. All rights reserved.

FILE 'WPIDS' ENTERED AT 07:44:19 ON 01 OCT 2004
COPYRIGHT (C) 2004 THE THOMSON CORPORATION

FILE 'USPATFULL' ENTERED AT 07:44:19 ON 01 OCT 2004
CA INDEXING COPYRIGHT (C) 2004 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOTECHDS' ENTERED AT 07:44:19 ON 01 OCT 2004
COPYRIGHT (C) 2004 THE THOMSON CORPORATION

FILE 'HCAPLUS' ENTERED AT 07:44:19 ON 01 OCT 2004
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2004 AMERICAN CHEMICAL SOCIETY (ACS)

=> s smurf activity adj2 regulat?
L1 0 SMURF ACTIVITY ADJ2 REGULAT?

=> s smurf activity
L2 2 SMURF ACTIVITY

=> d l2 ti abs ibib tot

L2 ANSWER 1 OF 2 WPIDS COPYRIGHT 2004 THE THOMSON CORP on STN
TI Novel isolated Smurf protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.
AN 2001-071267 [08] WPIDS
AB WO 200077168 A UPAB: 20011129
NOVELTY - An isolated Smurf1 or Smurf2 protein (I), is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) an isolated nucleic acid (II) encoding (I);
(2) a vector (III) comprising (II);
(3) a host cell (IV) comprising (III);
(4) production of (I);
(5) a transgenic non-human animal that expresses a human (I);
(6) screening (M) for a modulator of **Smurf activity**, comprising detecting modulation of **Smurf activity** in the presence of a test compound relative to **Smurf activity** in the absence of the test compound;
(7) an antibody (V) that specifically binds to (I);
(8) an oligonucleotide or nucleic acid (VI) that specifically hybridizes to (II) under highly stringent conditions; and
(9) promoting a bone morphogenic protein or transforming growth factor (TGF)- beta activation pathway in a cell, comprising suppressing expression of endogenous Smurf in the cell.
ACTIVITY - Cytostatic.
MECHANISM OF ACTION - Negative regulator of Smad signal transduction; antagonist of BMP and TGF- beta signaling pathway.
The inhibition of Smad1 by Smurf1 was tested. By over expressing Smad1 and Smad2 together with various dosages of Smurf1 in Xenopus animal caps, the ability of Smurf1 to directly antagonize the mesoderm induction

activities of Smad1 and Smad2, was tested. The results showed that expression of Smad1 alone induced ventral mesoderm, as demonstrated by expression of the ventral/posterior mesodermal markers Xhox3 and Xcad1. However, co-expression of Smurf1 and Smad1 blocked induction of these markers at all Smurf1 doses tested, demonstrating that Smurf1 can antagonize Smad1 activity.

USE - Expression of (I) from (III) in a cell is useful for inhibiting a bone morphogenic protein (BMP) or transforming growth factor- beta (TGF beta) activation pathway in a cell (claimed). (I) is useful to block chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. (I) is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it by antagonizing or mimicking the activity of (I), respectively, and in screening assays for identifying specific ligands of (I). (I) is useful as an immunogen to generate antibodies that are useful to alter the BMP pathway by inhibiting (I) or for diagnostic purposes. (I) is useful for treating a disorder associated with BMP or TGF- beta activation, such as cancer. (I) or inhibitor of (I) can be delivered by a vector to modulate Smads, e.g. to prevent Smurf regulation of Smads where BMP or TGF beta activity is desired, such as in bone regeneration or to study Smurf regulator processes in vivo.

Dwg.0/18

ACCESSION NUMBER: 2001-071267 [08] WPIDS
DOC. NO. CPI: C2001-019969
TITLE: Novel isolated Smurf protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.
DERWENT CLASS: B04 D16
INVENTOR(S): THOMSEN, G H; WRANA, J
PATENT ASSIGNEE(S): (HSCR-N) HSC RES & DEV LP; (UYNY) UNIV NEW YORK STATE RES FOUND
COUNTRY COUNT: 93
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000077168	A2	20001221	(200108)*	EN	106
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000056107	A	20010102	(200121)		
EP 1192174	A2	20020403	(200230)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
JP 2003502064	W	20030121	(200308)		131
CN 1409722	A	20030409	(200345)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000077168	A2	WO 2000-US16250	20000612
AU 2000056107	A	AU 2000-56107	20000612
EP 1192174	A2	EP 2000-941398	20000612
		WO 2000-US16250	20000612
JP 2003502064	W	WO 2000-US16250	20000612

		JP 2001-504003	20000612
CN 1409722	A	CN 2000-811354	20000612

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000056107	A	Based on	WO 2000077168
EP 1192174	A2	Based on	WO 2000077168
JP 2003502064	W	Based on	WO 2000077168

PRIORITY APPLN. INFO: US 1999-138969P 19990611

L2 ANSWER 2 OF 2 BIOTECHDS COPYRIGHT 2004 THE THOMSON CORP. on STN

TI Novel isolated Smurf protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation;
involving vector plasmid pCMV5-mediated gene transfer for expression in host cell

AN 2001-04474 BIOTECHDS

AB An isolated Smurf1 or Smurf2 protein (I), is claimed. Also claimed are: an isolated nucleic acid (II) encoding (I); a vector comprising (II); a host cell; production of (I); a transgenic non-human animal that expresses a human (I); screening for modulator of **Smurf activity**; an antibody that specifically binds to (I); an oligonucleotide or nucleic acid that specifically hybridizes to (II) under stringent conditions; and promoting a bone morphogenic protein or transforming growth factor (TGF)-beta activation pathway in a cell, comprising suppressing expression of endogenous Smurf in the cell. Expression of (I) from the vector in a cell is useful for inhibiting a bone morphogenic protein or TGF-beta activation pathway in a cell. (I) is useful to block chondrogenesis, osteogenesis, blood differentiation, cartilage formation, etc. (I) is useful for screening for various drugs and/or antibodies that can either enhance the bone morphogenic protein pathway, or inhibit it by antagonizing or mimicking the activity of (I), respectively. (I) is useful for treating a disorder associated with bone morphogenic protein or TGF-beta activation, such as cancer. (106pp)

ACCESSION NUMBER: 2001-04474 BIOTECHDS
TITLE: Novel isolated Smurf protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation;
involving vector plasmid pCMV5-mediated gene transfer for expression in host cell
AUTHOR: Thomsen G H; Wrana J
PATENT ASSIGNEE: Univ.New-York-State-Res.Found.; HSC-Res.Develop.
LOCATION: Toronto, Ontario, Canada.
PATENT INFO: WO 2000077168 21 Dec 2000
APPLICATION INFO: WO 2000-US16250 12 Jun 2000
PRIORITY INFO: US 1999-138969 11 Jun 1999
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2001-071267 [08]

=> d his

(FILE 'HOME' ENTERED AT 07:39:28 ON 01 OCT 2004)

FILE 'MEDLINE, BIOSIS, DGENE, EMBASE, WPIDS, USPATFULL, BIOTECHDS, HCAPLUS' ENTERED AT 07:44:19 ON 01 OCT 2004
L1 0 S SMURF ACTIVITY ADJ2 REGULAT?
L2 2 S SMURF ACTIVITY

=> s smurf
L3 104 SMURF

=> d l3 ti abs ibib tot

L3 ANSWER 1 OF 104 MEDLINE on STN
TI Transforming growth factor-beta stimulates p300-dependent RUNX3 acetylation, which inhibits ubiquitination-mediated degradation.
AB The Runt domain transcription factors (RUNXs) play essential roles in normal development and neoplasias. Genetic analyses of animals and humans have revealed the involvement of RUNX1 in hematopoiesis and leukemia, RUNX2 in osteogenesis and cleidocranial dysplasia, and RUNX3 in the development of T-cells and dorsal root ganglion neurons and in the genesis of gastric cancer. Here we report that RUNX3 is a target of the acetyltransferase activity of p300. The p300-dependent acetylation of three lysine residues protects RUNX3 from ubiquitin ligase **Smurf** -mediated degradation. The extent of the acetylation is up-regulated by the transforming growth factor-beta signaling pathway and down-regulated by histone deacetylase activities. Our findings demonstrate that the level of RUNX3 protein is controlled by the competitive acetylation and deacetylation of the three lysine residues, revealing a new mechanism for the posttranslational regulation of RUNX3 expression.
ACCESSION NUMBER: 2004349788 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15138260
TITLE: Transforming growth factor-beta stimulates p300-dependent RUNX3 acetylation, which inhibits ubiquitination-mediated degradation.
AUTHOR: Jin Yun-Hye; Jeon Eun-Joo; Li Qing-Lin; Lee Yong Hee; Choi Joong-Kook; Kim Wun-Jae; Lee Kwang-Youl; Bae Suk-Chul
CORPORATE SOURCE: Department of Biochemistry and Urology, School of Medicine and Institute for Tumor Research, Chungbuk National University, Cheongju 361-763, South Korea.
SOURCE: Journal of biological chemistry, (2004 Jul 9) 279 (28) 29409-17.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200408
ENTRY DATE: Entered STN: 20040716
Last Updated on STN: 20040825
Entered Medline: 20040824

L3 ANSWER 2 OF 104 MEDLINE on STN
TI Germline stem cell number in the Drosophila ovary is regulated by redundant mechanisms that control Dpp signaling.
AB The available experimental data support the hypothesis that the cap cells (CpCs) at the anterior tip of the germarium form an environmental niche for germline stem cells (GSCs) of the Drosophila ovary. Each GSC undergoes an asymmetric self-renewal division that gives rise to both a GSC, which remains associated with the CpCs, and a more posterior located cystoblast (CB). The CB upregulates expression of the novel gene, bag of marbles (bam), which is necessary for germline differentiation. Decapentaplegic (Dpp), a BMP2/4 homologue, has been postulated to act as a highly localized niche signal that maintains a GSC fate solely by repressing bam transcription. Here, we further examine the role of Dpp in GSC maintenance. In contrast to the above model, we find that an enhancer trap inserted near the Dpp target gene, Daughters against Dpp (Dad), is expressed in additional somatic cells within the germarium, suggesting that Dpp protein may be distributed throughout the anterior germarium. However, Dad-lacZ expression within the germline is present only in GSCs and to a lower level in CBs, suggesting there are mechanisms that actively restrict Dpp signaling in germ cells. We demonstrate that one function of

Bam is to block Dpp signaling downstream of Dpp receptor activation, thus establishing the existence of a negative feedback loop between the action of the two genes. Moreover, in females doubly mutant for bam and the ubiquitin protein ligase **Smurf**, the number of germ cells responsive to Dpp is greatly increased relative to the number observed in either single mutant. These data indicate that there are multiple, genetically redundant mechanisms that act within the germline to downregulate Dpp signaling in the Cb and its descendants, and raise the possibility that a Cb and its descendants must become refractory to Dpp signaling in order for germline differentiation to occur.

ACCESSION NUMBER: 2004207440 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15105369
TITLE: Germline stem cell number in the Drosophila ovary is regulated by redundant mechanisms that control Dpp signaling.
AUTHOR: Casanueva M Olivia; Ferguson Edwin L
CORPORATE SOURCE: Committee on Developmental Biology, University of Chicago, Chicago IL 60637, USA.
CONTRACT NUMBER: GM50838 (NIGMS)
SOURCE: Development (Cambridge, England), (2004 May) 131 (9) 1881-90.
Journal code: 8701744. ISSN: 0950-1991.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200407
ENTRY DATE: Entered STN: 20040424
Last Updated on STN: 20040703
Entered Medline: 20040702

L3 ANSWER 3 OF 104 MEDLINE on STN
TI Impaired Smad7-**Smurf**-mediated negative regulation of TGF-beta signaling in scleroderma fibroblasts.
AB The principal effect of TGF-beta1 on mesenchymal cells is its stimulation of ECM synthesis. Previous reports indicated the significance of the autocrine TGF-beta loop in the pathogenesis of scleroderma. In this study, we focused on Smad7 and Smurfs, principal molecules in the negative regulation of TGF-beta signaling, to further understand the autocrine TGF-beta loop in scleroderma. Scleroderma fibroblasts exhibited increased Smad7 levels compared with normal fibroblasts in vivo and in vitro. Smad7 constitutively formed a complex with the TGF-beta receptors, and the inhibitory effect of Smad7 on the promoter activity of human alpha2(I) collagen and 3TP-lux was completely impaired in scleroderma fibroblasts. Furthermore, the protein stability of TGF-beta receptor type I was significantly increased in scleroderma fibroblasts compared with normal fibroblasts. There was no significant difference in Smurf1 and Smurf2 levels between normal and scleroderma fibroblasts, and the transiently overexpressed Smurf1 and/or Smurf2 did not affect TGF-beta receptor type I protein levels in scleroderma fibroblasts. These results indicate that the impaired Smad7-**Smurf**-mediated inhibitory effect on TGF-beta signaling might contribute to maintaining the autocrine TGF-beta loop in scleroderma fibroblasts. To our knowledge, this is the first report of a disturbed negative regulation of TGF-beta signaling in fibrotic disorders.

ACCESSION NUMBER: 2004023363 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14722617
TITLE: Impaired Smad7-**Smurf**-mediated negative regulation of TGF-beta signaling in scleroderma fibroblasts.
AUTHOR: Asano Yoshihide; Ihn Hironobu; Yamane Kenichi; Kubo Masahide; Tamaki Kunihiko
CORPORATE SOURCE: Department of Dermatology, Faculty of Medicine, University of Tokyo, Tokyo, Japan.
SOURCE: Journal of clinical investigation, (2004 Jan) 113 (2) 253-64.

Journal code: 7802877. ISSN: 0021-9738.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200402
ENTRY DATE: Entered STN: 20040115
Last Updated on STN: 20040210
Entered Medline: 20040209

L3 ANSWER 4 OF 104 MEDLINE on STN
TI Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.
AB Smad ubiquitin regulatory factor (**Smurf**) 1 binds to receptor-regulated Smads for bone morphogenetic proteins (BMPs) Smad1/5 and promotes their degradation. In addition, Smurf1 associates with transforming growth factor-beta type I receptor through the inhibitory Smad (I-Smad) Smad7 and induces their degradation. Herein, we examined whether Smurf1 negatively regulates BMP signaling together with the I-Smads Smad6/7. Smurf1 and Smad6 cooperatively induced secondary axes in Xenopus embryos. Using a BMP-responsive promoter-reporter construct in mammalian cells, we found that Smurf1 cooperated with I-Smad in inhibiting BMP signaling and that the inhibitory activity of Smurf1 was not necessarily correlated with its ability to bind to Smad1/5 directly. Smurf1 bound to BMP type I receptors via I-Smads and induced ubiquitination and degradation of these receptors. Moreover, Smurf1 associated with Smad1/5 indirectly through I-Smads and induced their ubiquitination and degradation. Smurf1 thus controls BMP signaling with and without I-Smads through multiple mechanisms.
ACCESSION NUMBER: 2003328281 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12857866
TITLE: Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.
AUTHOR: Murakami Gyo; Watabe Tetsuro; Takaoka Kunio; Miyazono Kohei; Imamura Takeshi
CORPORATE SOURCE: Department of Biochemistry, The Cancer Institute of the Japanese Foundation for Cancer Research, Tokyo 170-8455, Japan.
SOURCE: Molecular biology of the cell, (2003 Jul) 14 (7) 2809-17. Journal code: 9201390. ISSN: 1059-1524.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200404
ENTRY DATE: Entered STN: 20030715
Last Updated on STN: 20040414
Entered Medline: 20040413

L3 ANSWER 5 OF 104 MEDLINE on STN
TI Cell cycle regulatory E3 ubiquitin ligases as anticancer targets.
AB Disregulation of the cell cycle and proliferation play key roles in cellular transformation and tumorigenesis. Such processes are intimately tied to the concentration, localization and activity of enzymes, adapters, receptors, and structural proteins in cells. Ubiquitination of these cellular regulatory proteins, governed by specific enzymes in the ubiquitin (Ub) conjugation cascade, has profound effects on their various functions, most commonly through proteasome targeting and degradation. This review will focus on a variety of E3 Ub ligases as potential oncology drug targets, with particular emphasis on the role of these molecules in the regulation of stability, localization, and activity of key proteins such as tumor suppressors and oncoproteins. E3 ubiquitin ligases that have established roles in cell cycle and apoptosis, such as the anaphase-promoting complex (APC), the Skp-1-Cul1-F-box class, and the

murine double minute 2 (MDM2) protein, in addition to more recently discovered E3 ubiquitin ligases which may be similarly important in tumorigenesis, (e.g. **Smurf** family, CHFR, and Efp), will be discussed. We will present evidence to support E3 ligases as good biological targets in the development of anticancer therapeutics and address challenges in drug discovery for these targets.

ACCESSION NUMBER: 2003024782 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12531181
TITLE: Cell cycle regulatory E3 ubiquitin ligases as anticancer targets.
AUTHOR: Pray Todd R; Parlati Francesco; Huang Jianing; Wong Brian R; Payan Donald G; Bennett Mark K; Issakani Sarkiz Daniel; Molineaux Susan; Demo Susan D
CORPORATE SOURCE: Rigel Pharmaceuticals, Inc., 240 East Grand Avenue, South San Francisco, California 94080, USA.. tpray@rigel.com
SOURCE: Drug resistance updates : reviews and commentaries in antimicrobial and anticancer chemotherapy, (2002 Dec) 5 (6) 249-58. Ref: 80
Journal code: 9815369. ISSN: 1368-7646.
PUB. COUNTRY: Scotland: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200305
ENTRY DATE: Entered STN: 20030118
Last Updated on STN: 20030521
Entered Medline: 20030520

L3 ANSWER 6 OF 104 MEDLINE on STN
TI Smad2 mediates transforming growth factor-beta induction of endothelial nitric oxide synthase expression.
AB Transforming growth factor-beta (TGF-beta) increases expression of endothelial nitric oxide synthase (eNOS), although the precise mechanism by which it does so is unclear. We report that Smad2, a transcription factor activated by TGF-beta, mediates TGF-beta induction of eNOS in endothelial cells. TGF-beta induces Smad2 translocation from cytoplasm to nucleus, where it directly interacts with a specific region of the eNOS promoter. Overexpression of Smad2 increases basal levels of eNOS, and further increases TGF-beta stimulation of eNOS expression. Ectopic expression of **Smurf**, an antagonizer of Smad2, decreases Smad2 expression and blocks TGF-beta induction of eNOS. Because Smad2 can interact with a variety of transcription factors, coactivators, and corepressors, Smad2 may thus act as an integrator of multiple signals in the regulation of eNOS expression.

ACCESSION NUMBER: 2002652771 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12411395
TITLE: Smad2 mediates transforming growth factor-beta induction of endothelial nitric oxide synthase expression.
AUTHOR: Saura Marta; Zaragoza Carlos; Cao Wangsen; Bao Clare; Rodriguez-Puyol Manuel; Rodriguez-Puyol Diego; Lowenstein Charles J
CORPORATE SOURCE: Department of Physiology, Universidad de Alcala, Madrid, Spain.
CONTRACT NUMBER: P01 HL56091 (NHLBI)
P01 HL65608 (NHLBI)
R01 HL53615 (NHLBI)
R01 HL63706 (NHLBI)
SOURCE: Circulation research, (2002 Nov 1) 91 (9) 806-13.
Journal code: 0047103. ISSN: 1524-4571.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 200211
ENTRY DATE: Entered STN: 20021105
Last Updated on STN: 20030304
Entered Medline: 20021105

L3 ANSWER 7 OF 104 MEDLINE on STN
TI The DSmurf ubiquitin-protein ligase restricts BMP signaling spatially and temporally during Drosophila embryogenesis.
AB We identified Drosophila **Smurf** (DSmurf) as a negative regulator of signaling by the BMP2/4 ortholog DPP during embryonic dorsal-ventral patterning. DSmurf encodes a HECT domain ubiquitin-protein ligase, homologous to vertebrate Smurf1 and Smurf2, that binds the Smad1/5 ortholog MAD and likely promotes its proteolysis. The essential function of DSmurf is restricted to its action on the DPP pathway. DSmurf has two distinct, possibly mechanistically separate, functions in controlling DPP signaling. Prior to gastrulation, DSmurf mutations cause a spatial increase in the DPP gradient, as evidenced by ventrolateral expansion in expression domains of target genes representing all known signaling thresholds. After gastrulation, DSmurf mutations cause a temporal delay in downregulation of earlier DPP signals, resulting in a lethal defect in hindgut organogenesis.
ACCESSION NUMBER: 2001654271 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11703946
TITLE: The DSmurf ubiquitin-protein ligase restricts BMP signaling spatially and temporally during Drosophila embryogenesis.
AUTHOR: Podos S D; Hanson K K; Wang Y C; Ferguson E L
CORPORATE SOURCE: Department of Molecular Genetics and Cell Biology, University of Chicago, Illinois 60637, USA.
CONTRACT NUMBER: GM50838 (NIGMS)
HD07959 (NICHD)
SOURCE: Developmental cell, (2001 Oct) 1 (4) 567-78.
Journal code: 101120028. ISSN: 1534-5807.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF416571
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011115
Last Updated on STN: 20030304
Entered Medline: 20011207

L3 ANSWER 8 OF 104 MEDLINE on STN
TI A new **Smurf** in the village.
AB TGF-beta signaling is modulated by Smurfs, E3-ubiquitin ligases that selectively target the receptors and Smad proteins for degradation. New evidence from Drosophila suggests that Smurfs regulate the amplitude and the duration of the cellular response to signaling in vivo.
ACCESSION NUMBER: 2001654258 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11703932
TITLE: A new **Smurf** in the village.
AUTHOR: Arora K; Warrior R
CORPORATE SOURCE: Department of Developmental and Cell Biology, University of California, Irvine 92697, USA.
SOURCE: Developmental cell, (2001 Oct) 1 (4) 441-2.
Journal code: 101120028. ISSN: 1534-5807.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011115
Last Updated on STN: 20020123

Entered Medline: 20011207

L3 ANSWER 9 OF 104 MEDLINE on STN
TI The hydrostatic and hydrodynamic volumes of polyols in aqueous solutions and their sweet taste.
AB The tastes and solution properties of sugar alcohols were studied in an attempt to illuminate the mechanism of sweet taste chemoreception. The **SMURF** method was used to measure tastetime-intensity of aqueous solutions of sugar alcohols and the results were interpreted using the Stevens power function and kinetic parameters. The apparent molar volumes, apparent specific volumes, partial molar volumes, partial specific volumes and intrinsic viscosities of the solutions were studied. Apparent molar volume reflects the size of the molecule in a hydrostatic state whereas intrinsic viscosity gives a measure of the size of the molecules in a hydrodynamic state. Generally the apparent molar volumes of the polyols are 6-13% greater than those of the parent sugars, indicating less interaction with the water structure. Apparent specific volume values can predict taste quality, and the average apparent specific volume for the sugar alcohols studied fits within the central part of the sweet range, i.e. 0.5-0.68 cm3/g, which accords with their ability to elicit a pure sweet taste response. Intensities and persistences of sweetness in the polyols followed the same trend as intrinsic viscosities.
ACCESSION NUMBER: 97292388 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9146905
TITLE: The hydrostatic and hydrodynamic volumes of polyols in aqueous solutions and their sweet taste.
AUTHOR: Lopez Chavez A; Birch G G
CORPORATE SOURCE: Department of Agriculture & Food Technology, ITESM, Queretaro, Mexico.
SOURCE: Chemical senses, (1997 Apr) 22 (2) 149-61.
Journal code: 8217190. ISSN: 0379-864X.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970805
Last Updated on STN: 19970805
Entered Medline: 19970723

L3 ANSWER 10 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Transforming growth factor-beta stimulates p300-dependent RUNX3 acetylation, which inhibits ubiquitination-mediated degradation.
AB The Runt domain transcription factors (RUNXs) play essential roles in normal development and neoplasias. Genetic analyses of animals and humans have revealed the involvement of RUNX1 in hematopoiesis and leukemia, RUNX2 in osteogenesis and cleidocranial dysplasia, and RUNX3 in the development of T-cells and dorsal root ganglion neurons and in the genesis of gastric cancer. Here we report that RUNX3 is a target of the acetyltransferase activity of p300. The p300-dependent acetylation of three lysine residues protects RUNX3 from ubiquitin ligase **Smurf** -mediated degradation. The extent of the acetylation is up-regulated by the transforming growth factor-beta signaling pathway and down-regulated by histone deacetylase activities. Our findings demonstrate that the level of RUNX3 protein is controlled by the competitive acetylation and deacetylation of the three lysine residues, revealing a new mechanism for the posttranslational regulation of RUNX3 expression.
ACCESSION NUMBER: 2004:347664 BIOSIS
DOCUMENT NUMBER: PREV200400349524
TITLE: Transforming growth factor-beta stimulates p300-dependent RUNX3 acetylation, which inhibits ubiquitination-mediated

degradation.

AUTHOR(S): Jin, Yun-Hye; Jeon, Eun-Joo; Lin, Qing-; Lee, Yong Hee; Choi, Joong-Kook; Kim, Wun-Jae; Lee, Kwang-Youl [Reprint Author]; Bae, Suk-Chul

CORPORATE SOURCE: Sch MedDept Biochem, Chungbuk Natl Univ, Cheongju, 361763, South Korea
ginsenoside@runx3.co.kr; scbae@med.chungbuk.ac.kr

SOURCE: Journal of Biological Chemistry, (July 9 2004) Vol. 279, No. 28, pp. 29409-29417. print.
CODEN: JBCHA3. ISSN: 0021-9258.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 18 Aug 2004
Last Updated on STN: 18 Aug 2004

L3 ANSWER 11 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

TI Germline stem cell number in the Drosophila ovary is regulated by redundant mechanisms that control Dpp signaling.

AB The available experimental data support the hypothesis that the cap cells (CpCs) at the anterior tip of the germarium form an environmental niche for germline stem cells (GSCs) of the Drosophila ovary. Each GSC undergoes an asymmetric self-renewal division that gives rise to both a GSC, which remains associated with the CpCs, and a more posterior located cystoblast (CB). The CB upregulates expression of the novel gene, bag of marbles (bam), which is necessary for germline differentiation. Decapentaplegic (Dpp), a BMP2/4 homologue, has been postulated to act as a highly localized niche signal that maintains a GSC fate solely by repressing bam transcription. Here, we further examine the role of Dpp in GSC maintenance. In contrast to the above model, we find that an enhancer trap inserted near the Dpp target gene, Daughters against Dpp (Dad), is expressed in additional somatic cells within the germarium, suggesting that Dpp protein may be distributed throughout the anterior germarium. However, Dad-lacZ expression within the germline is present only in GSCs and to a lower level in CBs, suggesting there are mechanisms that actively restrict Dpp signaling in germ cells. We demonstrate that one function of Bam is to block Dpp signaling downstream of Dpp receptor activation, thus establishing the existence of a negative feedback loop between the action of the two genes. Moreover, in females doubly mutant for bam and the ubiquitin protein ligase **Smurf**, the number of germ cells responsive to Dpp is greatly increased relative to the number observed in either single mutant. These data indicate that there are multiple, genetically redundant mechanisms that act within the germline to downregulate Dpp signaling in the Cb and its descendants, and raise the possibility that a Cb and its descendants must become refractory to Dpp signaling in order for germline differentiation to occur.

ACCESSION NUMBER: 2004:310387 BIOSIS

DOCUMENT NUMBER: PREV200400304879

TITLE: Germline stem cell number in the Drosophila ovary is regulated by redundant mechanisms that control Dpp signaling.

AUTHOR(S): Casanueva, M. Olivia; Ferguson, Edwin L. [Reprint Author]

CORPORATE SOURCE: Committee Dev Biol, Univ Chicago, Chicago, IL, 60637, USA
elfergus@midway.uchicago.edu

SOURCE: Development (Cambridge), (May 2004) Vol. 131, No. 9, pp. 1881-1890. print.
CODEN: DEVPED. ISSN: 0950-1991.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 7 Jul 2004
Last Updated on STN: 7 Jul 2004

L3 ANSWER 12 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

TI SMURFs: Standard monitoring units for the recruitment of temperate reef fishes.

AB I evaluated a standard monitoring unit for the recruitment of reef fishes (**SMURF**) as a tool for ascertaining spatial and temporal patterns of reef fish recruitment in central California, USA. SMURFs consisted of a 1.0 X 0.35 m dia. cylinder of fine mesh plastic grid that contained a folded section of larger mesh plastic grid. SMURFs collected new recruits of 20 species of fish with 92% of the individuals collected from 10 species, mostly rockfish (genus Sebastes). An experiment varying depth of SMURFs in the water column (surface, mid-depth, or bottom) showed that surface SMURFs collected the greatest diversity of species and significantly greater abundance for eight species, with two species having significantly greater abundance on mid-depth SMURFs and three species having significantly greater abundance on bottom SMURFs. A comparison of cumulated recruitment from SMURFs that varied in sampling frequency (removal of new recruits every 1-3, 7, or 28 days) suggested that increasing the time between sampling caused a significant decrease in recruitment estimates for some species but not for others. To determine how well temporal patterns of recruitment to SMURFs reflected patterns to nearby reefs, I compared within season temporal patterns of recruitment to SMURFs with that at nearby reefs, estimated by visual transect surveys conducted on scuba. Temporal patterns of recruitment to SMURFs were significantly and positively related to early recruitment on reefs for one group of benthic-algal associated rockfish species when diver surveys were lagged by 30 days (r=0.87) and for another group of canopy-algal associated rockfish species when lagged by 5 days (r=0.72). SMURFs appeared to be an effective and efficient method for indexing relative rates of delivery of competent juveniles for many temperate nearshore reef fishes.

ACCESSION NUMBER: 2004:175395 BIOSIS
DOCUMENT NUMBER: PREV200400176996
TITLE: SMURFs: Standard monitoring units for the recruitment of temperate reef fishes.
AUTHOR(S): Ammann, Arnold J. [Reprint Author]
CORPORATE SOURCE: NOAA Fisheries, 110 Shaffer Road, Santa Cruz, CA, 95060, USA
arnold.ammann@noaa.gov
SOURCE: Journal of Experimental Marine Biology and Ecology, (24 February 2004) Vol. 299, No. 2, pp. 135-154. print.
ISSN: 0022-0981 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Mar 2004
Last Updated on STN: 31 Mar 2004

L3 ANSWER 13 OF 104 BIOSIS on STN

TI The RING-H2 protein RNF11 is overexpressed in breast cancer and is a target of Smurf2 E3 ligase.

AB The breast cancer-associated T2A10 clone was originally isolated from a cDNA library enriched for tumour messenger ribonucleic acids. Our survey of 125 microarrayed primary tumour tissues using affinity purified polyclonal antibodies has revealed that corresponding protein is overexpressed in invasive breast cancer and is weakly expressed in kidney and prostate tumours. Now known as RNF11, the gene encodes a RING-H2 domain and a PY motif, both of which mediate protein-protein interactions. In particular, the PPPPY sequence of RNF11 PY motif is identical to that of Smad7, which has been shown to bind to WW domains of Smurf2, an E3 ubiquitin ligase that mediates the ubiquitination and degradation of the TGFbeta receptor complex. Using various mutants of RNF11 in GST pulldown and immunoprecipitation assays, we found that RNF11 interacts with Smurf2 through the PY motif, leading to ubiquitination of both proteins. Smurf2 plays an active role in the repression of TGFbeta signalling, and our data indicate that overexpression of RNF11, through its interaction with

Smurf2, can restore TGFbeta responsiveness in transfected cells.
ACCESSION NUMBER: 2004:96043 BIOSIS
DOCUMENT NUMBER: PREV200400097304
TITLE: The RING-H2 protein RNF11 is overexpressed in breast cancer and is a target of Smurf2 E3 ligase.
AUTHOR(S): Subramaniam, V.; Li, H.; Wong, M.; Kitching, R.; Attisano, L.; Wrana, J.; Zubovits, J.; Burger, A. M.; Seth, A. [Reprint Author]
CORPORATE SOURCE: Department of Laboratory Medicine and Pathobiology, University of Toronto, Toronto, ON, Canada arun.seth@utoronto.ca
SOURCE: British Journal of Cancer, (20 October 2003) Vol. 89, No. 8, pp. 1538-1544. print. ISSN: 0007-0920 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Feb 2004
Last Updated on STN: 18 Feb 2004

L3 ANSWER 14 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Impaired Smad7-**Smurf**-mediated negative regulation of TGF-beta signaling in scleroderma fibroblasts.
AB The principal effect of TGF-beta1 on mesenchymal cells is its stimulation of ECM synthesis. Previous reports indicated the significance of the autocrine TGF-beta loop in the pathogenesis of scleroderma. In this study, we focused on Smad7 and Smurfs, principal molecules in the negative regulation of TGF-beta signaling, to further understand the autocrine TGF-beta loop in scleroderma. Scleroderma fibroblasts exhibited increased Smad7 levels compared with normal fibroblasts in vivo and in vitro. Smad7 constitutively formed a complex with the TGF-beta receptors, and the inhibitory effect of Smad7 on the promoter activity of human alpha2(I) collagen and 3TP-lux was completely impaired in scleroderma fibroblasts. Furthermore, the protein stability of TGF-beta receptor type I was significantly increased in scleroderma fibroblasts compared with normal fibroblasts. There was no significant difference in Smurf1 and Smurf2 levels between normal and scleroderma fibroblasts, and the transiently overexpressed Smurf1 and/or Smurf2 did not affect TGF-beta receptor type I protein levels in scleroderma fibroblasts. These results indicate that the impaired Smad7-**Smurf**-mediated inhibitory effect on TGF-beta signaling might contribute to maintaining the autocrine TGF-beta loop in scleroderma fibroblasts. To our knowledge, this is the first report of a disturbed negative regulation of TGF-beta signaling in fibrotic disorders.
ACCESSION NUMBER: 2004:94938 BIOSIS
DOCUMENT NUMBER: PREV200400084043
TITLE: Impaired Smad7-**Smurf**-mediated negative regulation of TGF-beta signaling in scleroderma fibroblasts.
AUTHOR(S): Asano, Yoshihide; Ihn, Hironobu [Reprint Author]; Yamane, Kenichi; Kubo, Masahide; Tamaki, Kunihiko
CORPORATE SOURCE: Department of Dermatology, Faculty of Medicine, University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo, 113-8655, Japan IN-DER@h.u-tokyo.ac.jp
SOURCE: Journal of Clinical Investigation, (January 2004) Vol. 113, No. 2, pp. 253-264. print. CODEN: JCINAO. ISSN: 0021-9738.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Feb 2004
Last Updated on STN: 11 Feb 2004

L3 ANSWER 15 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.

AB Smad ubiquitin regulatory factor (**Smurf**) 1 binds to receptor-regulated Smads for bone morphogenetic proteins (BMPs) Smad1/5 and promotes their degradation. In addition, Smurf1 associates with transforming growth factor-beta type I receptor through the inhibitory Smad (I-Smad) Smad7 and induces their degradation. Herein, we examined whether Smurf1 negatively regulates BMP signaling together with the I-Smads Smad6/7. Smurf1 and Smad6 cooperatively induced secondary axes in Xenopus embryos. Using a BMP-responsive promoter-reporter construct in mammalian cells, we found that Smurf1 cooperated with I-Smad in inhibiting BMP signaling and that the inhibitory activity of Smurf1 was not necessarily correlated with its ability to bind to Smad1/5 directly. Smurf1 bound to BMP type I receptors via I-Smads and induced ubiquitination and degradation of these receptors. Moreover, Smurf1 associated with Smad1/5 indirectly through I-Smads and induced their ubiquitination and degradation. Smurf1 thus controls BMP signaling with and without I-Smads through multiple mechanisms.

ACCESSION NUMBER: 2003:356072 BIOSIS
DOCUMENT NUMBER: PREV200300356072
TITLE: Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.
AUTHOR(S): Murakami, Gyo; Watabe, Tetsuro; Takaoka, Kunio; Miyazono, Kohei [Reprint Author]; Imamura, Takeshi
CORPORATE SOURCE: Department of Biochemistry, The Cancer Institute of the Japanese Foundation for Cancer Research, Tokyo, 170-8455, Japan
miyazono-ind@umin.ac.jp
SOURCE: Molecular Biology of the Cell, (July 2003) Vol. 14, No. 7, pp. 2809-2817. print.
ISSN: 1059-1524 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Aug 2003
Last Updated on STN: 6 Aug 2003

L3 ANSWER 16 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

TI Smad2 mediates transforming growth factor-beta induction of endothelial nitric oxide synthase expression.

AB Transforming growth factor-beta (TGF-beta) increases expression of endothelial nitric oxide synthase (eNOS), although the precise mechanism by which it does so is unclear. We report that Smad2, a transcription factor activated by TGF-beta, mediates TGF-beta induction of eNOS in endothelial cells. TGF-beta induces Smad2 translocation from cytoplasm to nucleus, where it directly interacts with a specific region of the eNOS promoter. Overexpression of Smad2 increases basal levels of eNOS, and further increases TGF-beta stimulation of eNOS expression. Ectopic expression of **Smurf**, an antagonizer of Smad2, decreases Smad2 expression and blocks TGF-beta induction of eNOS. Because Smad2 can interact with a variety of transcription factors, coactivators, and corepressors, Smad2 may thus act as an integrator of multiple signals in the regulation of eNOS expression.

ACCESSION NUMBER: 2002:602932 BIOSIS
DOCUMENT NUMBER: PREV200200602932
TITLE: Smad2 mediates transforming growth factor-beta induction of endothelial nitric oxide synthase expression.
AUTHOR(S): Saura, Marta; Zaragoza, Carlos; Cao, Wangsen; Bao, Clare; Rodriguez-Puyol, Manuel; Rodriguez-Puyol, Diego; Lowenstein, Charles J. [Reprint author]
CORPORATE SOURCE: Division of Cardiology, Department of Medicine (W.C., C.B., C.J.L.), The Johns Hopkins University School of Medicine, Baltimore, MD, 21205, USA
clowenst@jhmi.edu
SOURCE: Circulation Research, (November 1, 2002) Vol. 91, No. 9, pp. 806-813. print.

CODEN: CIRUAL. ISSN: 0009-7330.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Nov 2002
Last Updated on STN: 27 Nov 2002

L3 ANSWER 17 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Specificity and complexity in **Smurf**-mediated Smad degradation.
ACCESSION NUMBER: 2002:133151 BIOSIS
DOCUMENT NUMBER: PREV200200133151
TITLE: Specificity and complexity in **Smurf**-mediated Smad degradation.
AUTHOR(S): Liang, Min [Reprint author]; Lin, Xia [Reprint author]; Liang, Yao-Yun [Reprint author]; Feng, Xin-Hua [Reprint author]; DeBakey, Michael E. [Reprint author]
CORPORATE SOURCE: Department of Surgery, Baylor College of Medicine, One Baylor Plaza, 139D, Houston, TX, 77030, USA
SOURCE: Molecular Biology of the Cell, (Nov, 2001) Vol. 12, No. Supplement, pp. 148a. print.
Meeting Info.: 41st Annual Meeting of the American Society for Cell Biology. Washington DC, USA. December 08-12, 2001. American Society for Cell Biology.
CODEN: MBCEEV. ISSN: 1059-1524.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Feb 2002
Last Updated on STN: 26 Feb 2002

L3 ANSWER 18 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Intracellular BMP signaling regulation in vertebrates: Pathway or network?.
AB Bone morphogenetic proteins (BMPs), members of the TGF-beta superfamily of secreted signaling molecules, have important functions in many biological contexts. They bind to specific serine/threonine kinase receptors, which transduce the signal to the nucleus through Smad proteins. The question of how BMPs can have such diverse effects while using the same canonical Smad pathway has recently come closer to an answer at the molecular level. Nuclear cofactors have been identified that cooperate with the Smads in regulating specific target genes depending on the cellular context. In addition, the pivotal role BMP signaling plays is underscored by the identification of factors that regulate members of this pathway at the cell surface, in the cytoplasm, and in the nucleus. Many of these factors are BMP-inducible and inhibit the BMP pathway, thus establishing negative feedback loops. Members of the BMP-Smad pathway can also physically interact with components of other signaling pathways to establish crosstalk. Finally, there is accumulating evidence that an alternative pathway involving MAP kinases can transduce BMP signals. The evidence and implications of these findings are discussed with an emphasis on early embryonic development of Xenopus and vertebrates.
ACCESSION NUMBER: 2001:540655 BIOSIS
DOCUMENT NUMBER: PREV200100540655
TITLE: Intracellular BMP signaling regulation in vertebrates: Pathway or network?.
AUTHOR(S): von Bubnoff, Andreas; Cho, Ken W. Y. [Reprint author]
CORPORATE SOURCE: Department of Developmental and Cell Biology, University of California, Irvine, CA, 92697-2300, USA
kwcho@uci.edu
SOURCE: Developmental Biology, (November 1, 2001) Vol. 239, No. 1, pp. 1-14. print.
CODEN: DEBIAO. ISSN: 0012-1606.
DOCUMENT TYPE: Article

General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Nov 2001
Last Updated on STN: 25 Feb 2002

L3 ANSWER 19 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Preliminary research on flora and vegetation in vertical karst shafts situated in the mountains strip of the Veneto pre-alps.
Original Title: Indagine preliminare su flora e vegetazione di pozzi carsici situati nella fascia montana delle Prealpi venete.
AB The study of the "Pozzo del Puffi" (**smurf**'s well) situated at 1580 mt. in the Grappa massif, is presented as an example of a research on flora and vegetation in the mountains strip of the Veneto pre-alps.After a morphological and ecological description, a flora list is discussed and an initial vegetation classification presented.
ACCESSION NUMBER: 2001:292272 BIOSIS
DOCUMENT NUMBER: PREV200100292272
TITLE: Preliminary research on flora and vegetation in vertical karst shafts situated in the mountains strip of the Veneto pre-alps.
Original Title: Indagine preliminare su flora e vegetazione di pozzi carsici situati nella fascia montana delle Prealpi venete.
AUTHOR(S): Faccio, Annamaria [Reprint author]; Busnardo, Giuseppe
CORPORATE SOURCE: Via della Concordia, 14, 36061, Bassano del Grappa, VI, Italy
SOURCE: Bollettino del Museo Civico di Storia Naturale di Venezia, (1998 (1999)) Vol. 49, No. Supplemento, pp. 373-380. print.
CODEN: BMSNAM. ISSN: 0505-205X.
DOCUMENT TYPE: Article
LANGUAGE: Italian
ENTRY DATE: Entered STN: 20 Jun 2001
Last Updated on STN: 19 Feb 2002

L3 ANSWER 20 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Arboreal beetles of neotropical forests: Agra Fabricius, the Novaurora complex (Coleoptera: Carabidae: Lebiini: Agrina).
AB The rufoaenea and quararibea groups (section Rufoaenea); the famula, formicaria, and phaenicodera groups (section Erythrnpus); and the capitata, cyanea, dimidiata, neblina, novaurora, and poguei groups constituted the study group for this paper because they share cribriform elytral intemeurs, an easily recognizable attribute for selecting specimens for study. They are referred to as the "Novaurora complex." The pusilla group, which shares interneur structural features with the Novaurora complex but little else, also was included in the key to groups. All of the above are treated in the key and are tersely described at the group level. The following groups are herein revised. The no novaurora group is a northern Amazon-Orinoco lineage comprising five species with a composite range extending from Ecuador to French Guiana and south into Brazil. Four specific taxa of the novaurora group are described as new (type locality in parentheses): alinahui (Ecuador: Napo Province, 20 km E Puerto Napo, Alinahui, 0degree10'S, 077degree25'W), orinocensis (Venezuela: Cano Marcareo, Orinoco Delta), novaurora (Ecuador: Napo province, 20 km E Puerto Napo, Alinahui, 01degree0'S, 077degree25'W), superba (Venezuela: T.F. Amazonas, confluence of Rio Negro and Rio Baria, 00degree55'N, 066degree10'W). The dimidiata group, predominantly northern Neotropical, comprises 16 species with a composite range extending from Mexico to northern Peru, and east to easternmost Venezuela. Thirteen specific taxa of the dimidiata group are described as new: bci (Panama: Barro Colorado Id., 09degree10'N, 079degree50'W), duckworthorum (Panama: Barro Colorado Id., 09degree10'N, 079degree50'W), eponine (Costa Rica: Puntarenas, Quepos, Parque Nacional Manuel Antonio, 09degree24'N,

08degree49'W), falcon (Venezuela: Falcon, Sanare, Finca Tillerias, 09degree39'N, 069degree45'W), hespenheide (Costa Rica: Heredia, La Selva, 10degree26'N, 084degree01'W), hovorei (Mexico: Vera Cruz, Estacion Biologica Los Tuxtlas, 18degree27'S, 095degree13'W), inbio (Costa Rica: Puntarenas, Mata de Limon, 09degree55'54"N, 084degree42'42"W), maracay (Venezuela: Maracay, 10degree15'N, 067degree36'W), parataz (Costa Rica: Puntarenas, Estacion Biologica Carara, E Quebrada Bonita, 09degree46'25"N, 084degree36'24"W), pichincha (Ecuador: Pichincha, Santo Domingo, Tinalandia, 00degree18'S, 079degree04'W), samiria (Peru: Loreto, Cocha Shinguito, 05degree08'S, 074degree45'W), tuxtlas (Mexico: Veracruz, Estacion Biologica Los Tuxtlas, near 18degree27'S, 095degree13'W), zapotal (Guatemala: Alta Verapaz, San Cristobal Verapaz, Quixal, 15degree23'N, 090degree24'W). The quararibea group is a southern and western Amazon-Pantanal lineage cornprising five species with a composite range extending from the upper Xingu drainage of Brazil west into Pern and Ecuador. Four specific taxa of the quararibea group are described as new: magnifica (Peru: Madre de Dios, 'Avispas" (Avispal), 12degree59'S, 071degree34'W), othello (Ecuador: Napo, 20 km E Puerto Napo, Alinahui, 01degree04'S, 077degree25'W), **smurf** (Brazil: Amazonas, Taperinha Santarem, 02degree32'S, 054degree17'W), suprerna (Brazil: Mato Grosso, Rosario Oeste, 14degree50'S, 056degree25'W). Distributions are dot-mapped and are discussed in general for each of the species in these three groups. Geographical ranges are given for all the groups of the Novaurora complex herein discussed.

ACCESSION NUMBER: 2000:303086 BIOSIS
DOCUMENT NUMBER: PREV200000303086
TITLE: Arboreal beetles of neotropical forests: Agra Fabricius, the Novaurora complex (Coleoptera: Carabidae: Lebiini: Agrina).
AUTHOR(S): Erwin, Terry L. [Reprint author]
CORPORATE SOURCE: Department of Entomology, National Museum of Natural History, Smithsonian Institution, Washington, DC, 20560-0169, USA
SOURCE: Smithsonian Contributions to Zoology, (2000) No. 608, pp. i-iii, 1-33. print.
CODEN: SMCZBU. ISSN: 0081-0282.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Jul 2000
Last Updated on STN: 7 Jan 2002

L3 ANSWER 21 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI Solute-solvent interactions and the sweet taste of small carbohydrates: Part II. Sweetness intensity and persistence in ethanol-water mixtures.
AB Intensity and persistence of sweet taste of sugars (glucose, fructose, xylose and sucrose) and polyols (sorbitol, xylitol) were determined in ethanol-water mixtures using a sensory measuring unit for recording flux (**SMURF**) device. In all cases sweetness intensity and persistence were decreased when ethanol concentration was increased from 10% to 30%. Assessing intensity/time responses for varied (from 2.3% to 9.2%, w/v) concentrations of D-glucose, D-fructose and sucrose in 5% ethanol mixture shows that persistence is more affected by the presence of ethanol than intensity. These results are interpreted by the solution properties in the ethanol-water binary solvent.

ACCESSION NUMBER: 1993:73437 BIOSIS
DOCUMENT NUMBER: PREV199395037937
TITLE: Solute-solvent interactions and the sweet taste of small carbohydrates: Part II. Sweetness intensity and persistence in ethanol-water mixtures.
AUTHOR(S): Hoopman, Tineke [Reprint author]; Birch, Gordon [Reprint author]; Serghat, Samira; Portmann, Marie-Odile; Mathlouthi, Mohamed
CORPORATE SOURCE: Dep. Food Sci. Technol., Univ. Reading, Whiteknights, PO

Box 226, Reading RG6 2AP, UK
SOURCE: Food Chemistry, (1993) Vol. 46, No. 2, pp. 147-153.
CODEN: FOCHDJ. ISSN: 0308-8146.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 26 Jan 1993
Last Updated on STN: 26 Jan 1993

L3 ANSWER 22 OF 104 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
TI STUDY OF SOME FACTORS AFFECTING INTENSITY-TIME CHARACTERISTICS OF SWEETNESS.
AB Intensity/time plots of sweetness produced by D-glucose, D-fructose and sucrose at concentrations ranging from 2.3 to 9.2% (w/v) were recorded for solutions at 15, 22 and 35° C. The intensity (I) and persistence (P) power functions were applied to the results obtained with a potentiometer connected to a chart recorder similar to the sensory measurement unit recording flux (**SMURF**) device. Increasing the concentration of assessed samples leads to an increase of perceived intensity with a tendency to show a compression for D-fructose and sucrose and an expansion of D-glucose. Persistence increases linearly as a function of concentration for the three sugars. Only very slight modification of intensity and persistence are observed when the temperature is varied from 15 to 35° C. Intensity/time plots were also recorded at 22° C for solutions containing 50% sucrose or equisweet concentrations of D-glucose or D-fructose brought to apparent viscosities of 5, 15, 25 and 35 mPa by addition of maltodextrins. It was found that the sweetness intensity decreases as viscosity increases for D-fructose and sucrose solutions whereas it remains constant for D-glucose. The persistence remains almost constant for the three sugars when the viscosity is varied. The effect of temperature on viscosity coefficients and hydration numbers is measured for the three sugars. A decrease in intrinsic viscosity [η], B-coefficients and hydration numbers is observed with increasing temperature whilst the apparent specific volume is increased. From the Raman spectra of water and aqueous solutions of sugars, it may be concluded that increasing the temperature leads to a lowering of the rigidity of the hydrogen bonded clusters and an increase in mobility of H2O molecules. The increase in the size of the sugars derived from apparent specific volume (.ovrhdot.VO2°) values reduces their accessibility to the receptor site. This effect is minimised as regards the perceived sweetness by the increased mobility of water. The effects of concentration, temperature and viscosity on the intensity and persistence of the sweet taste of D-glucose, D-fructose and sucrose, together with their physico-chemical properties in dilute solution, suggest that the accessibility of the sweet molecule to the receptor is an important step in the taste chemoreception. This step is followed by a biochemical phenomenon involving opening of ion channels which is sensitive to the mobility of water around the site and the sweetener.
ACCESSION NUMBER: 1992:279579 BIOSIS
DOCUMENT NUMBER: PREV199294004229; BA94:4229
TITLE: STUDY OF SOME FACTORS AFFECTING INTENSITY-TIME CHARACTERISTICS OF SWEETNESS.
AUTHOR(S): PORTMANN M-O [Reprint author]; SERGHAT S; MATHLOUTHI M
CORPORATE SOURCE: LAB CHIMIE PHYSIQUE INDUSTRIELLE, FAC SCI, UNIV REIMS-CHAMPAGNE-ARDENNE, BP 347, 51106 REIMS CEDEX, FR
SOURCE: Food Chemistry, (1992) Vol. 44, No. 2, pp. 83-92.
CODEN: FOCHDJ. ISSN: 0308-8146.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 10 Jun 1992
Last Updated on STN: 10 Jun 1992

L3 ANSWER 23 OF 104 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -

AN AAB31477 Protein DGENE
AB The present sequence represents a human Smurf2 polypeptide. The specification also describes a Smurf1 polypeptide. **Smurf** polypeptides are negative regulators of Smad signal transduction, and antagonists of bone morphogenic protein (BMP) or transforming growth factor-beta (TGF-beta) signalling pathway. Expression of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation pathway in a cell. **Smurf** polypeptides are useful for blocking chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. The polypeptide is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it.
ACCESSION NUMBER: AAB31477 Protein DGENE
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -
INVENTOR: Thomsen G H; Wrana J
PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.
(HSCR-N) HSC RES & DEV LP.
PATENT INFO: WO 2000077168 A2 20001221 107p
APPLICATION INFO: WO 2000-US16250 20000612
PRIORITY INFO: US 1999-138969 19990611
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-071267 [08]
CROSS REFERENCES: N-PSDB: AAF24853
DESCRIPTION: Amino acid sequence of a human Smurf2 polypeptide.

L3 ANSWER 24 OF 104 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -

AN AAB31476 Protein DGENE
AB The present sequence represents a human Smurf1 polypeptide. The specification also describes a Smurf2 polypeptide. **Smurf** polypeptides are negative regulators of Smad signal transduction, and antagonists of bone morphogenic protein (BMP) or transforming growth factor-beta (TGF-beta) signalling pathway. Expression of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation pathway in a cell. **Smurf** polypeptides are useful for blocking chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. The polypeptide is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it.
ACCESSION NUMBER: AAB31476 Protein DGENE
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -
INVENTOR: Thomsen G H; Wrana J
PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.

(HSCR-N) HSC RES & DEV LP.
PATENT INFO: WO 2000077168 A2 20001221 107p
APPLICATION INFO: WO 2000-US16250 20000612
PRIORITY INFO: US 1999-138969 19990611
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-071267 [08]
CROSS REFERENCES: N-PSDB: AAF24852
DESCRIPTION: Amino acid sequence of a human Smurf1 polypeptide.

L3 ANSWER 25 OF 104 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -

AN AAF24855 DNA DGENE
AB PCR primers AAF24854-55 were used to amplify human Smurf2 cDNA. The specification also describes a Smurf1 polypeptide. **Smurf** polypeptides are negative regulators of Smad signal transduction, and antagonists of bone morphogenic protein (BMP) or transforming growth factor-beta (TGF-beta) signalling pathway. Expression of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation pathway in a cell. **Smurf** polypeptides are useful for blocking chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. The polypeptide is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it.
ACCESSION NUMBER: AAF24855 DNA DGENE
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -
INVENTOR: Thomsen G H; Wrana J
PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.
(HSCR-N) HSC RES & DEV LP.
PATENT INFO: WO 2000077168 A2 20001221 107p
APPLICATION INFO: WO 2000-US16250 20000612
PRIORITY INFO: US 1999-138969 19990611
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-071267 [08]
DESCRIPTION: PCR primer used to amplify human Smurf1 cDNA.

L3 ANSWER 26 OF 104 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -

AN AAF24854 DNA DGENE
AB PCR primers AAF24854-55 were used to amplify human Smurf2 cDNA. The specification also describes a Smurf1 polypeptide. **Smurf** polypeptides are negative regulators of Smad signal transduction, and antagonists of bone morphogenic protein (BMP) or transforming growth factor-beta (TGF-beta) signalling pathway. Expression of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation pathway in a cell. **Smurf** polypeptides are useful for blocking chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the

BMP pathway. The polypeptide is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it.

ACCESSION NUMBER: AAF24854 DNA DGENE
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -
INVENTOR: Thomsen G H; Wrana J
PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.
(HSCR-N) HSC RES & DEV LP.
PATENT INFO: WO 2000077168 A2 20001221 107p
APPLICATION INFO: WO 2000-US16250 20000612
PRIORITY INFO: US 1999-138969 19990611
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-071267 [08]
DESCRIPTION: PCR primer used to amplify human Smurf1 cDNA.

L3 ANSWER 27 OF 104 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -
AN AAF24853 cDNA DGENE
AB The present sequence encodes a human Smurf2 polypeptide. The specification also describes a Smurf1 polypeptide. **Smurf** polypeptides are negative regulators of Smad signal transduction, and antagonists of bone morphogenic protein (BMP) or transforming growth factor-beta (TGF-beta) signalling pathway. Expression of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation pathway in a cell. **Smurf** polypeptides are useful for blocking chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. The polypeptide is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it.

ACCESSION NUMBER: AAF24853 cDNA DGENE
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -
INVENTOR: Thomsen G H; Wrana J
PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.
(HSCR-N) HSC RES & DEV LP.
PATENT INFO: WO 2000077168 A2 20001221 107p
APPLICATION INFO: WO 2000-US16250 20000612
PRIORITY INFO: US 1999-138969 19990611
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-071267 [08]
CROSS REFERENCES: P-PSDB: AAB31477
DESCRIPTION: Nucleotide sequence of a human Smurf2 polypeptide.

L3 ANSWER 28 OF 104 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -
AN AAF24852 cDNA DGENE
AB The present sequence encodes a human Smurf1 polypeptide. The specification also describes a Smurf2 polypeptide. **Smurf** polypeptides are negative regulators of Smad signal transduction, and

antagonists of bone morphogenic protein (BMP) or transforming growth factor-beta (TGF-beta) signalling pathway. Expression of Smurf1 in a cell is useful for inhibiting a BMP or TGF-beta activation pathway in a cell. **Smurf** polypeptides are useful for blocking chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. The polypeptide is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it.

ACCESSION NUMBER: AAF24852 cDNA DGENE
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation -
INVENTOR: Thomsen G H; Wrana J
PATENT ASSIGNEE: (UYNY)UNIV NEW YORK STATE RES FOUND.
(HSCR-N) HSC RES & DEV LP.
PATENT INFO: WO 2000077168 A2 20001221 107p
APPLICATION INFO: WO 2000-US16250 20000612
PRIORITY INFO: US 1999-138969 19990611
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-071267 [08]
CROSS REFERENCES: P-PSDB: AAB31476
DESCRIPTION: Nucleotide sequence of a human Smurf1 polypeptide.

L3 ANSWER 29 OF 104 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Transforming growth factor-β stimulates p300-dependent RUNX3 acetylation, which inhibits ubiquitination-mediated degradation.
AB The Runt domain transcription factors (RUNXs) play essential roles in normal development and neoplasias. Genetic analyses of animals and humans have revealed the involvement of RUNX1 in hematopoiesis and leukemia, RUNX2 in osteogenesis and cleidocranial dysplasia, and RUNX3 in the development of T-cells and dorsal root ganglion neurons and in the genesis of gastric cancer. Here we report that RUNX3 is a target of the acetyltransferase activity of p300. The p300-dependent acetylation of three lysine residues protects RUNX3 from ubiquitin ligase **Smurf** -mediated degradation. The extent of the acetylation is up-regulated by the transforming growth factor-β signaling pathway and down-regulated by histone deacetylase activities. Our findings demonstrate that the level of RUNX3 protein is controlled by the competitive acetylation and deacetylation of the three lysine residues, revealing a new mechanism for the posttranslational regulation of RUNX3 expression.

ACCESSION NUMBER: 2004300181 EMBASE
TITLE: Transforming growth factor-β stimulates p300-dependent RUNX3 acetylation, which inhibits ubiquitination-mediated degradation.
AUTHOR: Jin Y.-H.; Jeon E.-J.; Li Q.-L.; Lee Y.H.; Choi J.-K.; Kim W.-J.; Lee K.-Y.; Bae S.-C.
CORPORATE SOURCE: K.-Y. Lee, Department of Biochemistry, Sch. of Med. and Inst. for Tum. Res., Chungbuk National University, Cheongju 361-763, Korea, Republic of. ginsenoside@runx3.co.kr
SOURCE: Journal of Biological Chemistry, (9 Jul 2004) 279/28 (29409-29417).
Refs: 38
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 30 OF 104 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Germline stem cell number in the Drosophila ovary is regulated by redundant mechanisms that control Dpp signaling.

AB The available experimental data support the hypothesis that the cap cells (CpCs) at the anterior tip of the germarium form an environmental niche for germline stem cells (GSCs) of the Drosophila ovary. Each GSC undergoes an asymmetric self-renewal division that gives rise to both a GSC, which remains associated with the CpCs, and a more posterior located cystoblast (CB). The CB upregulates expression of the novel gene, bag of marbles (bam), which is necessary for germline differentiation. Decapentaplegic (Dpp), a BMP2/4 homologue, has been postulated to act as a highly localized niche signal that maintains a GSC fate solely by repressing bam transcription. Here, we further examine the role of Dpp in GSC maintenance. In contrast to the above model, we find that an enhancer trap inserted near the Dpp target gene, Daughters against Dpp (Dad), is expressed in additional somatic cells within the germarium, suggesting that Dpp protein may be distributed throughout the anterior germarium. However, Dad-lacZ expression within the germline is present only in GSCs and to a lower level in CBs, suggesting there are mechanisms that actively restrict Dpp signaling in germ cells. We demonstrate that one function of Bam is to block Dpp signaling downstream of Dpp receptor activation, thus establishing the existence of a negative feedback loop between the action of the two genes. Moreover, in females doubly mutant for bam and the ubiquitin protein ligase **Smurf**, the number of germ cells responsive to Dpp is greatly increased relative to the number observed in either single mutant. These data indicate that there are multiple, genetically redundant mechanisms that act within the germline to downregulate Dpp signaling in the Cb and its descendants, and raise the possibility that a Cb and its descendants must become refractory to Dpp signaling in order for germline differentiation to occur.

ACCESSION NUMBER: 2004237954 EMBASE
TITLE: Germline stem cell number in the Drosophila ovary is regulated by redundant mechanisms that control Dpp signaling.
AUTHOR: Casanueva M.O.; Ferguson E.L.
CORPORATE SOURCE: E.L. Ferguson, Committee on Developmental Biology, University of Chicago, Chicago, IL 60637, United States. elfergus@midway.uchicago.edu
SOURCE: Development, (2004) 131/9 (1881-1890).
Refs: 36
ISSN: 0950-1991 CODEN: DEVPED
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 021 Developmental Biology and Teratology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 31 OF 104 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Impaired Smad7-**Smurf**-mediated negative regulation of TGF-β signaling in scleroderma fibroblasts.

AB The principal effect of TGF-β on mesenchymal cells is its stimulation of ECM synthesis. Previous reports indicated the significance of the autocrine TGF-β loop in the pathogenesis of scleroderma. In this study, we focused on Smad7 and Smurfs, principal molecules in the negative regulation of TGF-β signaling, to further understand the autocrine TGF-β loop in scleroderma. Scleroderma fibroblasts exhibited increased Smad7 levels compared with normal fibroblasts in vivo and in vitro. Smad7 constitutively formed a complex with the TGF-β receptors, and the inhibitory effect of Smad7 on the promoter activity of human α2(1) collagen and 3TP-lux was completely impaired in

scleroderma fibroblasts. Furthermore, the protein stability of TGF-β receptor type I was significantly increased in scleroderma fibroblasts compared with normal fibroblasts. There was no significant difference in Smurf1 and Smurf2 levels between normal and scleroderma fibroblasts, and the transiently overexpressed Smurf1 and/or Smurf2 did not affect TGF-β receptor type I protein levels in scleroderma fibroblasts. These results indicate that the impaired Smad7-**Smurf**-mediated inhibitory effect on TGF-β signaling might contribute to maintaining the autocrine TGF-β loop in scleroderma fibroblasts. To our knowledge, this is the first report of a disturbed negative regulation of TGF-β signaling in fibrotic disorders.

ACCESSION NUMBER: 2004190200 EMBASE
TITLE: Impaired Smad7-**Smurf**-mediated negative regulation of TGF-β signaling in scleroderma fibroblasts.
AUTHOR: Asano Y.; Ihn H.; Yamane K.; Kubo M.; Tamaki K.
CORPORATE SOURCE: H. Ihn, Department of Dermatology, Faculty of Medicine, University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113-8655, Japan. 1N-DER@h.u-tokyo.ac.jp
SOURCE: Journal of Clinical Investigation, (2004) 113/2 (253-264).
Refs: 37
ISSN: 0021-9738 CODEN: JCINAO
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 32 OF 104 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.
AB Smad ubiquitin regulatory factor (**Smurf**) 1 binds to receptor-regulated Smads for bone morphogenetic proteins (BMPs) Smad1/5 and promotes their degradation. In addition, Smurf1 associates with transforming growth factor-β type I receptor through the inhibitory Smad (I-Smad) Smad7 and induces their degradation. Herein, we examined whether Smurf1 negatively regulates BMP signaling together with the I-Smads Smad6/7. Smurf1 and Smad6 cooperatively induced secondary axes in Xenopus embryos. Using a BMP-responsive promoter-reporter construct in mammalian cells, we found that Smurf1 cooperated with I-Smad in inhibiting BMP signaling and that the inhibitory activity of Smurf1 was not necessarily correlated with its ability to bind to Smad1/5 directly. Smurf1 bound to BMP type I receptors via I-Smads and induced ubiquitination and degradation of these receptors. Moreover, Smurf1 associated with Smad1/5 indirectly through I-Smads and induced their ubiquitination and degradation. Smurf1 thus controls BMP signaling with and without I-Smads through multiple mechanisms.

ACCESSION NUMBER: 2003293267 EMBASE
TITLE: Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads.
AUTHOR: Murakami G.; Watabe T.; Takaoka K.; Miyazono K.; Imamura T.
CORPORATE SOURCE: K. Miyazono, Department of Biochemistry, Cancer Inst. Japan. Found. Cancer R., Tokyo 170-8455, Japan. miyazono-ind@umin.ac.jp
SOURCE: Molecular Biology of the Cell, (1 Jul 2003) 14/7 (2809-2817).
Refs: 29
ISSN: 1059-1524 CODEN: MBCEEV
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 33 OF 104 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Cell cycle regulatory E3 ubiquitin ligases as anticancer targets.

AB Disregulation of the cell cycle and proliferation play key roles in cellular transformation and tumorigenesis. Such processes are intimately tied to the concentration, localization and activity of enzymes, adapters, receptors, and structural proteins in cells. Ubiquitination of these cellular regulatory proteins, governed by specific enzymes in the ubiquitin (Ub) conjugation cascade, has profound effects on their various functions, most commonly through proteasome targeting and degradation. This review will focus on a variety of E3 Ub ligases as potential oncology drug targets, with particular emphasis on the role of these molecules in the regulation of stability, localization, and activity of key proteins such as tumor suppressors and oncoproteins. E3 ubiquitin ligases that have established roles in cell cycle and apoptosis, such as the anaphase-promoting complex (APC), the Skp-1-Cu11-F-box class, and the murine double minute 2 (MDM2) protein, in addition to more recently discovered E3 ubiquitin ligases which may be similarly important in tumorigenesis, (e.g. **Smurf** family, CHFR, and Efp), will be discussed. We will present evidence to support E3 ligases as good biological targets in the development of anticancer therapeutics and address challenges in drug discovery for these targets. .COPYRGT. 2002 Elsevier Science Ltd. All rights reserved.

ACCESSION NUMBER: 2003048760 EMBASE

TITLE: Cell cycle regulatory E3 ubiquitin ligases as anticancer targets.

AUTHOR: Pray T.R.; Parlati F.; Huang J.; Wong B.R.; Payan D.G.; Bennett M.K.; Issakani S.D.; Molineaux S.; Demo S.D.

CORPORATE SOURCE: S.D. Demo, Rigel Pharmaceuticals, Inc., 240 East Grand Avenue, South San Francisco, CA 94080, United States. sdemo@rigel.com

SOURCE: Drug Resistance Updates, (2002) 5/6 (249-258).
Refs: 81
ISSN: 1368-7646 CODEN: DRUPFW

PUBLISHER IDENT.: S 1368-7646(02)00121-8

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 016 Cancer
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

L3 ANSWER 34 OF 104 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Smad2 mediates transforming growth factor-β induction of endothelial nitric oxide synthase expression.

AB Transforming growth factor-β (TGF-β) increases expression of endothelial nitric oxide synthase (eNOS), although the precise mechanism by which it does so is unclear. We report that Smad2, a transcription factor activated by TGF-β, mediates TGF-β induction of eNOS in endothelial cells. TGF-β induces Smad2 translocation from cytoplasm to nucleus, where it directly interacts with a specific region of the eNOS promoter. Overexpression of Smad2 increases basal levels of eNOS, and further increases TGF-β stimulation of eNOS expression. Ectopic expression of **Smurf**, an antagonizer of Smad2, decreases Smad2 expression and blocks TGF-β induction of eNOS. Because Smad2 can interact with a variety of transcription factors, coactivators, and corepressors, Smad2 may thus act as an integrator of multiple signals in the regulation of eNOS expression.

ACCESSION NUMBER: 2002402412 EMBASE

TITLE: Smad2 mediates transforming growth factor-β induction of endothelial nitric oxide synthase expression.

AUTHOR: Saura M.; Zaragoza C.; Cao W.; Bao C.; Rodriguez-Puyol M.;

Rodriguez-Puyol D.; Lowenstein C.J.
CORPORATE SOURCE: C.J. Lowenstein, Division of Cardiology, Department of Medicine, Johns Hopkins Univ. Sch. of Medicine, Baltimore, MD 21205, United States. clowenst@jhmi.edu
SOURCE: Circulation Research, (1 Nov 2002) 91/9 (806-813).
Refs: 62
ISSN: 0009-7330 CODEN: CIRUAL
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 35 OF 104 EMBASE on STN
TI Extracellular regulation of BMP signaling in vertebrates: A cocktail of modulators.
AB The transforming growth factor-β (TGF-β) superfamily contains a variety of growth factors which all share common sequence elements and structural motifs. These proteins are known to exert a wide spectrum of biological responses on a large variety of cell types in both vertebrates and invertebrates. Many of them have important functions during embryonic development in pattern formation and tissue specification, and in adult tissues, they are involved in processes such as wound healing, bone repair, and bone remodeling. The family is divided into two general branches: the BMP/GDF and the TGF-β/Activin/Nodal branches, whose members have diverse, often complementary effects. It is obvious that an orchestered regulation of different actions of these proteins is necessary for proper functioning. The TGF-β family members act by binding extracellularly to a complex of serine/threonine kinase receptors, which consequently activate Smad molecules by phosphorylation. These Smads translocate to the nucleus, where they modulate transcription of specific genes. Three levels by which this signaling pathway is regulated could be distinguished. First, a control mechanism exists in the intracellular space, where inhibitory Smads and Smurfs prevent further signaling and activation of target genes. Second, at the membrane site, the pseudoreceptor BAMBI/Nma is able to inhibit further signaling within the cells. Finally, a range of extracellular mediators are identified which modulate the functioning of members of the TGF-β superfamily. Here, we review the insights in the extracellular regulation of members of the BMP subfamily of secreted growth factors with a major emphasis on vertebrate BMP modulation. .COPYRGT. 2002 Elsevier Science (USA).
ACCESSION NUMBER: 2002378732 EMBASE
TITLE: Extracellular regulation of BMP signaling in vertebrates: A cocktail of modulators.
AUTHOR: Balemans W.; Van Hul W.
CORPORATE SOURCE: W. Van Hul, Department of Medical Genetics, University of Antwerp, University Hospital, Antwerp 2610, Belgium. vhul@uia.ac.be
SOURCE: Developmental Biology, (2002) 250/2 (231-250).
Refs: 181
ISSN: 0012-1606 CODEN: DEBIAO
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 021 Developmental Biology and Teratology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 36 OF 104 EMBASE on STN
TI TGF-β induces assembly of a Smad2-Smurf2 ubiquitin ligase complex that targets SnoN for degradation.

AB The receptor-regulated Smad proteins are essential intracellular mediators of signal transduction by the transforming growth factor-β (TGF-β) superfamily of growth factors and are also important as regulators of gene transcription. Here we describe a new role for TGF-β-regulated Smad2 and Smad3 as components of a ubiquitin ligase complex. We show that in the presence of TGF-β signalling, Smad2 interacts through its proline-rich PPXY motif with the tryptophan-rich WW domains of Smurf2, a recently identified E3 ubiquitin ligases. TGF-β also induces the association of Smurf2 with the transcriptional co-repressor SnoN and we show that Smad2 can function to mediate this interaction. This allows Smurf2 HECT domain to target SnoN for ubiquitin-mediated degradation by the proteasome. Thus, stimulation by TGF-β can induce the assembly of a Smad2-Smurf2 ubiquitin ligase complex that functions to target substrates for degradation.

ACCESSION NUMBER: 2001211670 EMBASE
TITLE: TGF-β induces assembly of a Smad2-Smurf2 ubiquitin ligase complex that targets SnoN for degradation.
AUTHOR: Bonni S.; Wang H.-R.; Causing C.G.; Kavsak P.; Stroschein S.L.; Luo K.; Wrana J.L.
CORPORATE SOURCE: J.L. Wrana, Program in Molecular Biology/Cancer, Samuel Lunenfeld Research Institute, Mount Sinai Hospital, 600 University Avenue, Toronto, Ont. M5G 1X5, Canada. wrana@mshri.on.ca
SOURCE: Nature Cell Biology, (2001) 3/6 (587-595).
Refs: 39
ISSN: 1465-7392 CODEN: NCBIFN
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 37 OF 104 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI The hydrostatic and hydrodynamic volumes of polyols in aqueous solutions and their sweet taste.
AB The tastes and solution properties of sugar alcohols were studied in an attempt to illuminate the mechanism of sweet taste chemoreception. The **SMURF** method was used to measure taste time-intensity of aqueous solutions of sugar alcohols and the results were interpreted using the Stevens power function and kinetic parameters. The apparent molar volumes, apparent specific volumes, partial molar volumes, partial specific volumes and intrinsic viscosities of the solutions were studied. Apparent molar volume reflects the size of the molecule in a hydrostatic state whereas intrinsic viscosity gives a measure of the size of the molecules in a hydrodynamic state. Generally the apparent molar volumes of the polyols are 6-13% greater than those of the parent sugars, indicating less interaction with the water structure. Apparent specific volume values can predict taste quality, and the average apparent specific volume for the sugar alcohols studied fits within the central part of the sweet range, i.e. 0.5-0.68 cm3/g, which accords with their ability to elicit a pure sweet taste response. Intensities and persistences of sweetness in the polyols followed the same trend as intrinsic viscosities.

ACCESSION NUMBER: 97119783 EMBASE
DOCUMENT NUMBER: 1997119783
TITLE: The hydrostatic and hydrodynamic volumes of polyols in aqueous solutions and their sweet taste.
AUTHOR: Chavez A.L.; Birch G.G.
CORPORATE SOURCE: A.L. Chavez, Dept. Agriculture Food Technology, ITESM-CQ, Apdo. Postal 37, Queretaro, Mexico
SOURCE: Chemical Senses, (1997) 22/2 (149-161).
Refs: 42
ISSN: 0379-864X CODEN: CHSED8
COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
029 Clinical Biochemistry
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 38 OF 104 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Use of the '**SMURF**' in taste analysis.
AB An improved moving chart recording of intensity/time of taste responses has been achieved using a potentiometer 'dial box' linked by a cable to a Telsec recorder. The device allows rates of taste response to be determined and is described as a Sensory Measuring Unit for Recording Flux (**SMURF**) on the assumption that the flux of stimuli at the taste receptor is responsible for the time course of response. Fourteen trained and sixteen untrained panellists evaluated one standard and four unknown sucrose solutions using the **SMURF** and determined their intensity and persistence time of response for each of these same solutions by conventional interval scaling and use of a stop-clock. The **SMURF** gave results which were higher (but not significantly so) than the conventional method. Trained panellists tended to prefer the **SMURF** and found it quicker and easier to use than the conventional method. Untrained panellists tended to prefer the conventional method but these results were generally not significant. The **SMURF** is therefore an extremely useful device in reducing time and effort whilst still maintaining accuracy in the measurement of intensity and time of taste response. The **SMURF** was also used to obtained intensity/time data for three other sugars so that a comparison between the sugars could be made.
ACCESSION NUMBER: 81166883 EMBASE
DOCUMENT NUMBER: 1981166883
TITLE: Use of the '**SMURF**' in taste analysis.
AUTHOR: Birch G.G.; Munton S.L.
CORPORATE SOURCE: Nat. Coll. Food Technol., Univ. Reading, Weybridge, Surrey, KT13 ODE, United Kingdom
SOURCE: Chemical Senses, (1981) 6/1 (45-52).
CODEN: CHSED8
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 002 Physiology
037 Drug Literature Index
LANGUAGE: English

L3 ANSWER 39 OF 104 WPIDS COPYRIGHT 2004 THE THOMSON CORP on STN
TI Network protection method for detecting denial of service attack, involves re-reading configuration file and terminating data structure using signal thread, and transmitting statistics associated with Radix trees using status thread.
AN 2004-604520 [58] WPIDS
AB WO2004070547 A UPAB: 20040910
NOVELTY - A data structure is initialized and system thread is activated by configuration thread. A packet is processed and trigger condition is updated by packet capture thread. Attack response is stopped by bookkeeper thread if trigger condition is not satisfied. A configuration file is re-read and data structure is terminated using signal thread, and statistics associated with Radix trees, is transmitted using status thread.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for network protection system.

USE - For protecting network such as Internet from harmful data traffic intended to data port attacks such as denial of service (DoS) attack e.g. buffer overflow attack, SYN attack, ping of death attack, teardrop attack, **smurf** attack, and other attacks such as

routing-based attack and unauthorized access to certain protected services in computer system.

ADVANTAGE - Allows each thread to execute other threads independently, thereby improving performance. Each thread shares to same data space with other threads, resulting in simplified inter-process communication.

DESCRIPTION OF DRAWING(S) - The figure shows a block diagram of the network protection system.

Dwg.1/8

ACCESSION NUMBER: 2004-604520 [58] WPIDS
DOC. NO. NON-CPI: N2004-478181
TITLE: Network protection method for detecting denial of service attack, involves re-reading configuration file and terminating data structure using signal thread, and transmitting statistics associated with Radix trees using status thread.
DERWENT CLASS: T01
INVENTOR(S): BILLQUIST, P G; GARBUTT, G W; NADLER, M H; SHANKLIN, C; SODMAN, D M
PATENT ASSIGNEE(S): (CAPT-N) CAPTUS NETWORKS CORP
COUNTRY COUNT: 108
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004070547	A2	20040819	(200458)*	EN	47

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004070547	A2	WO 2004-US2610	20040130

PRIORITY APPLN. INFO: US 2003-444571P 20030203

L3 ANSWER 40 OF 104 WPIDS COPYRIGHT 2004 THE THOMSON CORP on STN
TI Identifying protein-protein interactions, useful e.g. in drug development, comprises introducing into cells one or more prey proteins labeled with an epitope tag and one or more bait proteins labeled with a detectable substance.
AN 2004-315601 [29] WPIDS
AB WO2004023146 A UPAB: 20040505

NOVELTY - Identifying protein-protein interactions comprising prey proteins interacting with one or more bait comprises introducing one or more prey proteins in labeled with an epitope tag and one or more bait protein in cells labeled with a detectable substance.

DETAILED DESCRIPTION - Identifying protein-protein interactions comprising prey proteins interacting with one or more bait comprises:

(a) introducing one or more prey proteins in cells, where a prey is labeled with an epitope tag permitting separation of the prey protein from other proteins in the cells;

(b) introducing one or more bait protein in cells, where a bait protein is labeled with a detectable substance permitting detection of the bait protein and protein-protein interactions comprising a prey protein and the bait protein;

(c) inducing formation of protein-protein interactions between a prey

and bait protein; and

(d) assaying for protein-protein interactions comprising a prey protein and bait protein by detecting the detectable substance.

INDEPENDENT CLAIMS are also included for:

(1) quantitating protein-protein interactions;

(2) determining an interactome for one or more bait protein;

(3) determining the functions of gene product;

(4) systematically and quantitatively analyzing protein-protein interactions in cell signaling;

(5) determining the changes in an interactome of mitotic kinase during cell cycle progression;

(6) analyzing protein-protein interactions in different cell types;

(7) assaying for changes in protein-protein interactions in response to intracellular and extracellular factors;

(8) identifying a potential modulator of signal transduction activity; and

(9) an agent, modulator or inhibitor identified by a method of (8).

ACTIVITY - Antiinflammatory; Cytostatic.

No biological data given.

MECHANISM OF ACTION - None Given.

USE - The method and kits are useful in identifying, quantifying and analyzing protein-protein interactions. The method is useful in determining a disease or condition associated with a test protein, monitoring the course of therapy, conducting a drug discovery business and in detecting mutations in cellular proteins. The pharmaceutical composition is useful in treating and preventing a disease or condition associated with an abnormality in a signal transduction pathway, e.g. fibrosis, inflammation or cancer.

Dwg.0/3

ACCESSION NUMBER: 2004-315601 [29] WPIDS
DOC. NO. NON-CPI: N2004-251489
DOC. NO. CPI: C2004-119632
TITLE: Identifying protein-protein interactions, useful e.g. in drug development, comprises introducing into cells one or more prey proteins labeled with an epitope tag and one or more bait proteins labeled with a detectable substance.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): BARRIOS-RODILES, M; WRANA, J
PATENT ASSIGNEE(S): (MOUN) MOUNT SINAI HOSPITAL
COUNTRY COUNT: 105
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004023146	A2	20040318	(200429)*	EN	53
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
AU 2003264211	A1	20040329	(200459)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004023146	A2	WO 2003-CA1354	20030905
AU 2003264211	A1	AU 2003-264211	20030905

FILING DETAILS:

PATENT NO	KIND	PATENT NO

AU 2003264211 A1 Based on WO 2004023146

PRIORITY APPLN. INFO: US 2002-408922P 20020906

L3 ANSWER 41 OF 104 WPIDS COPYRIGHT 2004 THE THOMSON CORP on STN
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.
AN 2001-071267 [08] WPIDS
AB WO 200077168 A UPAB: 20011129
NOVELTY - An isolated Smurf1 or Smurf2 protein (I), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated nucleic acid (II) encoding (I);
(2) a vector (III) comprising (II);
(3) a host cell (IV) comprising (III);
(4) production of (I);
(5) a transgenic non-human animal that expresses a human (I);
(6) screening (M) for a modulator of **Smurf** activity, comprising detecting modulation of **Smurf** activity in the presence of a test compound relative to **Smurf** activity in the absence of the test compound;
(7) an antibody (V) that specifically binds to (I);
(8) an oligonucleotide or nucleic acid (VI) that specifically hybridizes to (II) under highly stringent conditions; and
(9) promoting a bone morphogenic protein or transforming growth factor (TGF)- beta activation pathway in a cell, comprising suppressing expression of endogenous **Smurf** in the cell.

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - Negative regulator of Smad signal transduction; antagonist of BMP and TGF- beta signaling pathway.

The inhibition of Smad1 by Smurf1 was tested. By over expressing Smad1 and Smad2 together with various dosages of Smurf1 in Xenopus animal caps, the ability of Smurf1 to directly antagonize the mesoderm induction activities of Smad1 and Smad2, was tested. The results showed that expression of Smad1 alone induced ventral mesoderm, as demonstrated by expression of the ventral/posterior mesodermal markers Xhox3 and Xcad1. However, co-expression of Smurf1 and Smad1 blocked induction of these markers at all Smurf1 doses tested, demonstrating that Smurf1 can antagonize Smad1 activity.

USE - Expression of (I) from (III) in a cell is useful for inhibiting a bone morphogenic protein (BMP) or transforming growth factor- beta (TGF beta) activation pathway in a cell (claimed). (I) is useful to block chondrogenesis, osteogenesis, blood differentiation, cartilage formation, neural tube patterning, retinal development, heart induction and morphogenesis, hair growth, tooth formation, gamete formation and a wide variety of tissue and organ formation processes, and hinder the regeneration, growth, maintenance, etc., of bone and other tissues that are dependent on the BMP pathway. (I) is useful for screening for various drugs and/or antibodies that can either enhance the BMP pathway, or inhibit it by antagonizing or mimicking the activity of (I), respectively, and in screening assays for identifying specific ligands of (I). (I) is useful as an immunogen to generate antibodies that are useful to alter the BMP pathway by inhibiting (I) or for diagnostic purposes. (I) is useful for treating a disorder associated with BMP or TGF- beta activation, such as cancer. (I) or inhibitor of (I) can be delivered by a vector to modulate Smads, e.g. to prevent **Smurf** regulation of Smads where BMP or TGF beta activity is desired, such as in bone regeneration or to study **Smurf** regulator processes in vivo.

Dwg.0/18

ACCESSION NUMBER: 2001-071267 [08] WPIDS
DOC. NO. CPI: C2001-019969
TITLE: Novel isolated **Smurf** protein useful for

inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation.

DERWENT CLASS: B04 D16

INVENTOR(S): THOMSEN, G H; WRANA, J

PATENT ASSIGNEE(S): (HSCR-N) HSC RES & DEV LP; (UYNY) UNIV NEW YORK STATE RES FOUND

COUNTRY COUNT: 93

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000077168	A2	20001221	(200108)*	EN	106
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000056107	A	20010102	(200121)		
EP 1192174	A2	20020403	(200230)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
JP 2003502064	W	20030121	(200308)		131
CN 1409722	A	20030409	(200345)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000077168	A2	WO 2000-US16250	20000612
AU 2000056107	A	AU 2000-56107	20000612
EP 1192174	A2	EP 2000-941398	20000612
		WO 2000-US16250	20000612
JP 2003502064	W	WO 2000-US16250	20000612
		JP 2001-504003	20000612
CN 1409722	A	CN 2000-811354	20000612

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000056107	A	Based on	WO 2000077168
EP 1192174	A2	Based on	WO 2000077168
JP 2003502064	W	Based on	WO 2000077168

PRIORITY APPLN. INFO: US 1999-138969P 19990611

L3 ANSWER 42 OF 104 USPATFULL on STN

TI Secure self-organizing and self-provisioning anomalous event detection systems

AB An approach for providing managed security services is disclosed. A database, within a server or a pre-existing anomalous event detection system, stores a rule set specifying a security policy for a network associated with a customer. An anomalous detection event module is deployed within a premise of the customer and retrieves rule sets from the database. The anomalous detection event module monitors a sub-network of the network based on the rule sets. The anomalous event detection module is further configured to self-organize by examining components of the network and to monitor for anomalous events according to the examined components, and to self-provision by selectively creating another instance of the anomalous detection event module to monitor another sub-network of the network.

ACCESSION NUMBER: 2004:234588 USPATFULL
TITLE: Secure self-organizing and self-provisioning anomalous event detection systems
INVENTOR(S): Hoefelmeyer, Ralph Samuel, Colorado Springs, CO, UNITED STATES
Phillips, Theresa E., Fairfax, VA, UNITED STATES
Wiederin, Shawn Edward, Cedar Rapids, IA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004181664	A1	20040916
APPLICATION INFO.:	US 2003-385229	A1	20030310 (10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WORLDCOM, INC., Technology Law Department, 1133 19th Street, N.W., Washington, DC, 20036
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 889

L3 ANSWER 43 OF 104 USPATFULL on STN
TI Method and system for managing of denial of service attacks using bandwidth allocation technology
AB A method and system for managing attacks in a computer system is disclosed. The computer system is used in sending, receiving, or sending and receiving a plurality of packets, which include a plurality of administrative packets. The method and system include determining whether a congestion of the administrative packets exists. Congestion of the administrative packets indicates that a potential attack exists. The method and system also include discarding a portion of the plurality of administrative packets if it is declared that the congestion of the administrative packets exists. The portion of the plurality of packets is sufficient to ensure that a remaining portion of the plurality of packets transmitted is not more than a maximum administrative packet bandwidth limit and, if the plurality of administrative packets present a sufficient offered load, not less than a minimum administrative packet bandwidth guarantee.

ACCESSION NUMBER: 2004:220359 USPATFULL
TITLE: Method and system for managing of denial of service attacks using bandwidth allocation technology
INVENTOR(S): Carpenter, Brian E., Kilchberg, SWITZERLAND
Jeffries, Clark D., Durham, NC, UNITED STATES
Kind, Andreas, Kilchberg, SWITZERLAND
Siegel, Michael S., Raleigh, NC, UNITED STATES
PATENT ASSIGNEE(S): International Business Machines Corporation, Armonk, NY (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004170123	A1	20040902
APPLICATION INFO.:	US 2003-375799	A1	20030227 (10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: IBM CORPORATION, PO BOX 12195, DEPT 9CCA, BLDG 002, RESEARCH TRIANGLE PARK, NC, 27709
NUMBER OF CLAIMS: 33
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 1099

L3 ANSWER 44 OF 104 USPATFULL on STN
TI Internet privacy protection device

AB The invention consists of a standalone broadband plug and play Internet privacy protection device that provides complete computer or network security for always-on high speed connections by means of combining a real-time packet inspection process in conjunction with computer or network IP address concealment and implementing a seamless network disconnection upon detection of Internet inactivity by the client.

ACCESSION NUMBER: 2004:210555 USPATFULL
TITLE: Internet privacy protection device
INVENTOR(S): Sami, Vikash Krishna, Burnaby, CANADA
Paraskake, Michael, Vancouver, CANADA

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004162992	A1	20040819	
APPLICATION INFO.:	US 2003-364322	A1	20030219	(10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Mr. Paul Prade, SAAFNET INTERNATIONAL INC., 5945 Kathleen Avenue, 6th Floor, Burnaby, British Columbia, V5H 4 J7
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 1606

L3 ANSWER 45 OF 104 USPATFULL on STN
TI Multilayered intrusion detection system and method
AB A multilayered intrusion detection system and method are disclosed. The method includes monitoring activity on a network and maintaining a registry of each host node address associated with a host node operable to perform host-based intrusion detection services. The method further includes comparing a destination address of the monitored network activity with at least one host node address in the registry. If an address of the network activity matches an address of a registered host node, the network activity is dismissed and allowed to proceed unencumbered to the registered host node. The network activity not destined for a registered host node has intrusion detection services performed on it. The network activity dismissed to the host node has intrusion detection services performed on it at the receiving host node.

ACCESSION NUMBER: 2004:200079 USPATFULL
TITLE: Multilayered intrusion detection system and method
INVENTOR(S): Baker, Stephen M., San Antonio, TX, United States
PATENT ASSIGNEE(S): Cisco Technology, Inc., San Jose, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6775657	B1	20040810	
APPLICATION INFO.:	US 1999-471508		19991222	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Starks, Jr., Wilbert L.
ASSISTANT EXAMINER: Booker, Kelvin
LEGAL REPRESENTATIVE: Baker Botts L.L.P.
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 1085

L3 ANSWER 46 OF 104 USPATFULL on STN
TI Method and apparatus for permitting visualizing network data
AB Methods and apparatuses for the visualization of network traffic and

permitting access thereto are provided. In one aspect of the invention, an illustrative method includes defining a plurality of views of network traffic for the classification of network traffic into the views. At least one of the views is a group view. In one example, the types of views include at least two of the following: network address, application, protocol, flow type, packet type, geographic region, ICMP type, slow scan, operating system, flag, remote host count, local host count, spoofing, fragments, service, sessions, response time, status, and user. In another example, network traffic is classified according to the composite views of various combinations of previously defined views. A master console permits users to access only the portion of the network for which the users is responsible. The permitted view does not show other parts of the network.

ACCESSION NUMBER: 2004:185771 USPATFULL
TITLE: Method and apparatus for permitting visualizing network data
INVENTOR(S): Newton, Chris, Douglas, CANADA
Bird, William, Estey's Bridge, CANADA
Spencer, Dwight, Douglas, CANADA

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004143658	A1	20040722	
APPLICATION INFO.:	US 2003-346920	A1	20030117	(10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: James C. Scheller, Jr., BLAKELY, SOKOLOFF, TAYLOR & ZAFMAN LLP, Seventh Floor, 12400 Wilshire Boulevard, Los Angeles, CA, 90025-1026
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 439

L3 ANSWER 47 OF 104 USPATFULL on STN
TI Detecting a network attack
AB In general, in one aspect, the disclosure describes techniques of detecting a network attack. The method includes receiving at least one packet at a device; and determining whether the at least one received packet has at least one characteristic of a denial of service attack. Based on the determining, the packet may not be processed by a transport layer protocol.

ACCESSION NUMBER: 2004:160337 USPATFULL
TITLE: Detecting a network attack
INVENTOR(S): Dubal, Scott P., Hillsboro, OR, UNITED STATES
Boom, Douglas D., Portland, OR, UNITED STATES
Connor, Patrick L., Portland, OR, UNITED STATES
Montecalvo, Mark V., Hillsboro, OR, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004123142	A1	20040624	
APPLICATION INFO.:	US 2002-323985	A1	20021218	(10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BLAKELY SOKOLOFF TAYLOR & ZAFMAN, 12400 WILSHIRE BOULEVARD, SEVENTH FLOOR, LOS ANGELES, CA, 90025
NUMBER OF CLAIMS: 38
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 423

L3 ANSWER 48 OF 104 USPATFULL on STN
TI Protection against denial of service attacks
AB An information processing system for protecting against denial of service attacks comprises an interface (310) to receive and send packets, wherein the packets comprise at least one synchronization packet that is part of a handshake process for establishing the connection between the source client computer (118) and the target server computer (102); a crypto engine (306) adapted to create a unique sequence number for inclusion in a packet to be sent to a client (118) requesting establishment of a connection between a client (118) and server (102), wherein the crypto engine (306) is further adapted to validate unique sequence numbers in received synchronization packets that are part of a handshake process for establishing the connection between the source client (118) and the protected server (102); and a lookup table (304) for storing information defining established connections between the server (102) and clients so that arriving packets that purport to be part of an established connection can be validated by comparing information in the packet with entries in the table.

ACCESSION NUMBER:	2004:145959 USPATFULL			
TITLE:	Protection against denial of service attacks			
INVENTOR(S):	Boivie, Richard Harold, Monroe, CT, UNITED STATES Fong, Jun Tung, Pleasantville, NY, UNITED STATES			
PATENT ASSIGNEE(S):	International Business Machines Corporation, Armonk, NY, UNITED STATES (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004111635	A1	20040610	
APPLICATION INFO.:	US 2002-308605	A1	20021204	(10)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	Michael J. Buchenhorner Esq., P.A., 1430 Sorolla Avenue, Coral Gables, FL, 33134			
NUMBER OF CLAIMS:	21			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	4 Drawing Page(s)			
LINE COUNT:	735			

L3 ANSWER 49 OF 104 USPATFULL on STN
TI Specification-based anomaly detection
AB A method for network intrusion detection on a network comprising a plurality of state machines for passing a plurality of network packets comprises determining frequency distributions for each transition within each state machine, determining the distributions of values of each state machine on each transition, and comparing the distributions to observed statistics in the network, and upon determining that the observed statistics are outside defined limits, detecting an anomaly.

ACCESSION NUMBER:	2004:128631 USPATFULL			
TITLE:	Specification-based anomaly detection			
INVENTOR(S):	Sekar, Ramasubramanian, East Setauket, NY, UNITED STATES			
PATENT ASSIGNEE(S):	Research Foundation of the State University of New York (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004098617	A1	20040520	
APPLICATION INFO.:	US 2002-298826	A1	20021118	(10)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	Frank Chau, F. CHAU & ASSOCIATES, LLP, Suite 501, 1900			

Hempstead Turnpike, East Meadow, NY, 11554
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 1033

L3 ANSWER 50 OF 104 USPATFULL on STN
TI Method and system to collect geographic location information for a network address utilizing geographically dispersed data collection agents
AB A method and a system perform geolocation activities relating to a network address. A database of network addresses, and associated geographic locations, is maintained. A query, including a network address, is received against the database for a geographic location associated with the network address. Information, concerning the query received against the database, is logged. Geolocation activities relating to at least the network address are modified based on the logged information.

ACCESSION NUMBER: 2004:102619 USPATFULL
TITLE: Method and system to collect geographic location information for a network address utilizing geographically dispersed data collection agents
INVENTOR(S): Anderson, Mark, Westminster, CO, UNITED STATES
Bansal, Ajay, San Jose, CA, UNITED STATES
Doctor, Brad, Broomfield, CO, UNITED STATES
Hadjiyiannis, George, Boston, MA, UNITED STATES
Herringshaw, Christopher, West Wardsboro, VT, UNITED STATES
Karplus, Eli E., Heidelberg, GERMANY, FEDERAL REPUBLIC OF
Muniz, Derald, Midlothian, TX, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004078490	A1	20040422
APPLICATION INFO.:	US 2003-686135	A1	20031014 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-825675, filed on 3 Apr 2001, GRANTED, Pat. No. US 6684250

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-194761P	20000403 (60)
	US 2000-241776P	20001018 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BLAKELY SOKOLOFF TAYLOR & ZAFMAN, 12400 WILSHIRE BOULEVARD, SEVENTH FLOOR, LOS ANGELES, CA, 90025
NUMBER OF CLAIMS: 48
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 64 Drawing Page(s)
LINE COUNT: 3160

L3 ANSWER 51 OF 104 USPATFULL on STN
TI Method and system to associate a geographic location information with a network address using a combination of automated and manual process
AB A method and a system map a geographic location to a network address. At least one automated process is performed to identify a geographic location for the network address. A determination is made whether the automated process provided satisfactory geographic location information for the network address. If the automated process did not provided satisfactory geographic location information for the network address, then the network address is forwarded for manual resolution.

ACCESSION NUMBER: 2004:102618 USPATFULL
TITLE: Method and system to associate a geographic location information with a network address using a combination of automated and manual process
INVENTOR(S): Anderson, Mark, Westminster, CO, UNITED STATES
Bansal, Ajay, San Jose, CA, UNITED STATES
Doctor, Brad, Broomfield, CO, UNITED STATES
Hadjiyiannis, George, Boston, MA, UNITED STATES
Herringshaw, Christopher, West Wardsboro, VT, UNITED STATES
Karplus, Eli E., Heidelberg, GERMANY, FEDERAL REPUBLIC OF
Muniz, Derald, Midlothian, TX, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004078489	A1	20040422
APPLICATION INFO.:	US 2003-685692	A1	20031014 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-825675, filed on 3 Apr 2001, GRANTED, Pat. No. US 6684250

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-194761P	20000403 (60)
	US 2000-241776P	20001018 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BLAKELY SOKOLOFF TAYLOR & ZAFMAN, 12400 WILSHIRE BOULEVARD, SEVENTH FLOOR, LOS ANGELES, CA, 90025
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 64 Drawing Page(s)
LINE COUNT: 3114

L3 ANSWER 52 OF 104 USPATFULL on STN
TI Method and system to modify geolocation activities based on logged query information
AB A method and a system perform geolocation activities relating to a network address. A database of network addresses, and associated geographic locations, is maintained. A query, including a network address, is received against the database for a geographic location associated with the network address. Information, concerning the query received against the database, is logged. Geolocation activities relating to at least the network address are modified based on the logged information.

ACCESSION NUMBER: 2004:102496 USPATFULL
TITLE: Method and system to modify geolocation activities based on logged query information
INVENTOR(S): Anderson, Mark, Westminister, CO, UNITED STATES
Bansal, Ajay, San Jose, CA, UNITED STATES
Doctor, Brad, Broomfield, CO, UNITED STATES
Hadjiyiannis, George, Boston, MA, UNITED STATES
Herringshaw, Christopher, West Wardsboro, VT, UNITED STATES
Karplus, Eli E., Heidelberg, GERMANY, FEDERAL REPUBLIC OF
Muniz, Derald, Midlothian, TX, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004078367	A1	20040422
APPLICATION INFO.:	US 2003-685991	A1	20031014 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-825675, filed on 3 Apr

2001, GRANTED, Pat. No. US 6684250

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-194761P	20000403 (60)
	US 2000-241776P	20001018 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BLAKELY SOKOLOFF TAYLOR & ZAFMAN, 12400 WILSHIRE BOULEVARD, SEVENTH FLOOR, LOS ANGELES, CA, 90025
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 64 Drawing Page(s)
LINE COUNT: 3168

L3 ANSWER 53 OF 104 USPATFULL on STN
TI Method and system to initiate geolocation activities on demand and responsive to receipt of a query
AB A method and the system perform geolocation activities relating to a network address. A query, including a network address, is received from an external entity at a geolocation system. Responsive to receipt of the query, geolocation activities are initiated at the geolocation system to map the network address to a geographic location.

ACCESSION NUMBER: 2004:89604 USPATFULL
TITLE: Method and system to initiate geolocation activities on demand and responsive to receipt of a query
INVENTOR(S): Anderson, Mark, Westminster, CO, UNITED STATES
Bansal, Ajay, San Jose, CA, UNITED STATES
Doctor, Brad, Broomfield, CO, UNITED STATES
Hadjiyiannis, George, Boston, MA, UNITED STATES
Herringshaw, Christopher, West Wardsboro, VT, UNITED STATES
Karplus, Eli E., Heidelberg, GERMANY, FEDERAL REPUBLIC OF
Muniz, Derald, Midlothian, TX, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004068582	A1	20040408
APPLICATION INFO.:	US 2003-686102	A1	20031014 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-825675, filed on 3 Apr 2001, GRANTED, Pat. No. US 6684250

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-194761P	20000403 (60)
	US 2000-241776P	20001018 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BLAKELY SOKOLOFF TAYLOR & ZAFMAN, 12400 WILSHIRE BOULEVARD, SEVENTH FLOOR, LOS ANGELES, CA, 90025
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 64 Drawing Page(s)
LINE COUNT: 3092

L3 ANSWER 54 OF 104 USPATFULL on STN
TI System and method for detecting and countering a network attack
AB Protecting a host network from a flood-type denial of service attack by performing statistical analysis of data packets in the network. The statistical analysis comprises comparing evaluated items in the data packets to threshold values and detecting the attack when the statistical items exceed the threshold value. A countermeasure can be

initiated to protect the host network from the attack.

ACCESSION NUMBER: 2004:71696 USPATFULL
TITLE: System and method for detecting and countering a network attack
INVENTOR(S): Etheridge, James K., Jupiter, FL, UNITED STATES
Anton, Richard N., Jupiter, FL, UNITED STATES
PATENT ASSIGNEE(S): Cyber Operations, LLC, Jupiter, FL, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004054925	A1	20040318
APPLICATION INFO.:	US 2002-243631	A1	20020913 (10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KING & SPALDING, 191 PEACHTREE STREET, N.E., ATLANTA, GA, 30303-1763
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 1021

L3 ANSWER 55 OF 104 USPATFULL on STN
TI Methods and materials for transformation
AB Disclosed herein are novel methods and materials directed to transforming a host cell and expressing exogenous RNA therein. Specifically disclosed are DNA-launching platforms used to introduce a replicating viral segment attached to an exogenous polynucleotide into a cell, whereby the exogenous polynucleotide is expressed in said cell and confers a detectable trait.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:59043 USPATFULL
TITLE: Methods and materials for transformation
INVENTOR(S): Rasochova, Lada, Madison, WI, UNITED STATES
German, Thomas, Hollandale, WI, UNITED STATES
Ahlquist, Paul, Madison, WI, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004045050	A1	20040304
APPLICATION INFO.:	US 2003-609207	A1	20030626 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1999-316622, filed on 21 May 1999, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-86526P	19980522 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SALIWANCHIK LLOYD & SALIWANCHIK, A PROFESSIONAL ASSOCIATION, 2421 N.W. 41ST STREET, SUITE A-1, GAINESVILLE, FL, 326066669
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 2817
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 56 OF 104 USPATFULL on STN
TI Architecture for high speed class of service enabled linecard
AB A linecard architecture for high speed routing of data in a communications device. This architecture provides low latency routing

based on packet priority: packet routing and processing occurs at line rate (wire speed) for most operations. A packet data stream is input to the inbound receiver, which uses a small packet FIFO to rapidly accumulate packet bytes. Once the header portion of the packet is received, the header alone is used to perform a high speed routing lookup and packet header modification. The queue manager then uses the class of service information in the packet header to enqueue the packet according to the required priority. Enqueued packets are buffered in a large memory space holding multiple packets prior to transmission across the device's switch fabric to the outbound linecard. On arrival at the outbound linecard, the packet is enqueued in the outbound transmitter portion of the linecard architecture. Another large, multi-packet memory structure, as employed in the inbound queue manager, provides buffering prior to transmission onto the network.

ACCESSION NUMBER:	2004:27457 USPATFULL
TITLE:	Architecture for high speed class of service enabled linecard
INVENTOR(S):	Wilford, Bruce, Los Altos, CA, United States Dan, Yie-Fong, Cupertino, CA, United States
PATENT ASSIGNEE(S):	Cisco Technology, Inc., San Jose, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6687247	B1	20040203	
APPLICATION INFO.:	US 1999-428870		19991027	(9)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	GRANTED
PRIMARY EXAMINER:	Pham, Chi
ASSISTANT EXAMINER:	Boakye, Alexander O.
LEGAL REPRESENTATIVE:	Campbell Stephenson Ascolese LLP
NUMBER OF CLAIMS:	41
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	33 Drawing Figure(s); 31 Drawing Page(s)
LINE COUNT:	3234

L3 ANSWER 57 OF 104 USPATFULL on STN

TI Method and apparatus for incrementally deploying ingress filtering on the internet

AB Ingress filtering has been adopted by the IETF as a methodology for preventing denial of service congestive attacks that spoof the source address in packets that are addressed to host server victims. Unless universally adopted by all ISPs on the Internet, however, a packet's source address cannot be totally trusted to be its actual source address. To take advantage of benefits of ingress filtering as it is gradually deployed by ISPs around the Internet, differentiated classes of service are used to transport packets whose source address can be trusted and packets whose source address cannot be trusted. A packet received by an access or edge router at an ISP that supports ingress filtering and has a source address that is properly associated with port on which it is received is forwarded in a privileged class of service and are dropped otherwise. A packet received by access or edge router at an ISP that does not support ingress filtering and whose source address cannot therefore be trusted is transported in an unprivileged class of service. At an intermediate exchange router within an intermediate ISP, where ISPs exchange packets, a packet received from an ISP that doesn't support ingress filtering is forwarded using the unprivileged class of service while a packet received from an ISP that does support ingress filtering is forwarded using the same class of service in which it is already marked.

ACCESSION NUMBER:	2003:336119 USPATFULL
TITLE:	Method and apparatus for incrementally deploying

ingress filtering on the internet
INVENTOR(S): Brustoloni, Jose?apos, C., Westfield, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003236999	A1	20031225
APPLICATION INFO.:	US 2002-175577	A1	20020619 (10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Lucent Technologies Inc., Docket Administrator (Room 3J-219), 101 Crawfords Corner Road, Holmdel, NJ, 07733-3030
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 557

L3 ANSWER 58 OF 104 USPATFULL on STN
TI Method and apparatus for facilitating detection of network intrusion
AB System for facilitating detection of network intrusion. Through continuous accumulation of network traffic parameter information, data for a particular session is reduced to a single metric that represents the threat potential of the session as compared to normal network traffic. An analysis station accumulates and maintains the historical data and defines a point for each specific session within a distribution. The dimensions in the distribution space take into account various network traffic parameters useful in identifying an attack. The distance between a session's point and the centroid of the distribution represents the threat metric. The analysis station can display the threat metric as a point or points on a display. The intensity of the point is an indication of the threat potential. The easy-to-read display calls anomalous traffic to the attention of an operator and facilitates discrimination among ambiguous cases.

ACCESSION NUMBER: 2003:336115 USPATFULL
TITLE: Method and apparatus for facilitating detection of network intrusion
INVENTOR(S): Fretwell, Lyman Jefferson, JR., Randolph, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003236995	A1	20031225
APPLICATION INFO.:	US 2002-177078	A1	20020621 (10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: STEVEN B. PHILLIPS, MOORE & VAN ALLEN, 2200 WEST MAIN STREET, SUITE 800, DURHAM, NC, 27705
NUMBER OF CLAIMS: 70
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Page(s)
LINE COUNT: 1896

L3 ANSWER 59 OF 104 USPATFULL on STN
TI Light activated gene transduction using long wavelength ultraviolet light for cell targeted gene delivery
AB In accordance with the present invention, methods are provided for treating a patient through the use of ultraviolet light activated gene therapy. Embodiments of the present invention include methods for the utilization of light activated gene therapy to repair and/or rebuild damaged cartilage by introducing a desired gene into a patient's tissue.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:335516 USPATFULL

TITLE: Light activated gene transduction using long wavelength ultraviolet light for cell targeted gene delivery

INVENTOR(S): Schwarz, Edward M., Rochester, NY, UNITED STATES
O'Keefe, Regis J., Pittsford, NY, UNITED STATES
Foster, Thomas, Rochester, NY, UNITED STATES
Finlay, Jarod C., Philadelphia, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003236394	A1	20031225
APPLICATION INFO.:	US 2003-357271	A1	20030131 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-353842P	20020131 (60)
	US 2002-353907P	20020131 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KNOBBE MARTENS OLSON & BEAR LLP, 2040 MAIN STREET, FOURTEENTH FLOOR, IRVINE, CA, 92614
NUMBER OF CLAIMS: 75
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 785
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 60 OF 104 USPATFULL on STN
TI Detecting randomness in computer network traffic
AB A method, system and computer program product for detecting denial-of-service attacks. The randomness in the Internet Protocol (IP) source addresses of transmitted IP packets may be detected by performing a hash function on the IP source addresses thereby generating one or more different hash values. If a high number of different hash values were generated for a small number of IP packets evaluated, then random IP source addresses may be detected. By detecting random source IP addresses, a denial-of-service attack may be detected.

ACCESSION NUMBER: 2003:284096 USPATFULL
TITLE: Detecting randomness in computer network traffic
INVENTOR(S): Jeffries, Clark Debs, Durham, NC, UNITED STATES
Jong, Wuchieh James, Raleigh, NC, UNITED STATES
Randall, Grayson Warren, Cary, NC, UNITED STATES
Vu, Ken Van, Cary, NC, UNITED STATES
PATENT ASSIGNEE(S): International Business Machines Corporation, Armonk, NY, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003200441	A1	20031023
APPLICATION INFO.:	US 2002-127031	A1	20020419 (10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: IBM CORPORATION, PO BOX 12195, DEPT 9CCA, BLDG 002, RESEARCH TRIANGLE PARK, NC, 27709
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 823

L3 ANSWER 61 OF 104 USPATFULL on STN
TI System and method for detecting an infective element in a network environment
AB A method for detecting an infective element in a network environment is provided that includes detecting, by a first computer, an infective

element within a second computer. A signal is generated and communicated that identifies the second computer as being associated with the infective element. The signal includes an address associated with the second computer. The signal is received and, in response to the signal, a communicating capability of the second computer is disabled.

ACCESSION NUMBER: 2003:272358 USPATFULL
TITLE: System and method for detecting an infective element in a network environment
INVENTOR(S): Gleichauf, Robert E., Terrell Hills, TX, UNITED STATES
PATENT ASSIGNEE(S): Cisco Technology, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003191966	A1	20031009
APPLICATION INFO.:	US 2002-119934	A1	20020409 (10)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BAKER BOTTS L.L.P., 2001 ROSS AVENUE, SUITE 600, DALLAS, TX, 75201-2980
NUMBER OF CLAIMS: 55
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 1084

L3 ANSWER 62 OF 104 USPATFULL on STN
TI Novel methods of diagnosis of angiogenesis, compositions and methods of screening for angiogenesis modulators
AB Described herein are methods and compositions that can be used for diagnosis and treatment of angiogenic phenotypes and angiogenesis-associated diseases. Also described herein are methods that can be used to identify modulators of angiogenesis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:219636 USPATFULL
TITLE: Novel methods of diagnosis of angiogenesis, compositions and methods of screening for angiogenesis modulators
INVENTOR(S): Murray, Richard, Cupertino, CA, UNITED STATES
Glynne, Richard, Palo Alto, CA, UNITED STATES
Watson, Susan R., El Cerrito, CA, UNITED STATES
PATENT ASSIGNEE(S): Eos Biotechnology, Inc., South San Francisco, CA, UNITED STATES, 94080 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003152926	A1	20030814
APPLICATION INFO.:	US 2001-21660	A1	20011206 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2001-784356, filed on 14 Feb 2001, PENDING Continuation-in-part of Ser. No. US 2000-637977, filed on 11 Aug 2000, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-148425P	19990811 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
LINE COUNT: 10887
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 63 OF 104 USPATFULL on STN
TI Method for representing, storing and editing network security policy
AB A network security policy is represented, stored and edited by using a rule object, a condition object, an action object, and their associations. The condition object is a one-packet-condition object, a repeated-packet-condition object or a linear-packet-condition object. The action object is an alert-action object, a packet-drop-action object, a packet-admission-action object, a session-drop-action object, a session-admission-action object, a session-logging-action object, a traceback-action object or an ICMP-unreachable-message-sending-action object.

ACCESSION NUMBER: 2003:195947 USPATFULL
TITLE: Method for representing, storing and editing network security policy
INVENTOR(S): Kim, Sook Yeon, Daejeon, KOREA, REPUBLIC OF
Kim, Geon Lyang, Jeollanam-do, KOREA, REPUBLIC OF
Kim, Myung Eun, Daejeon, KOREA, REPUBLIC OF
Kim, Ki Young, Daejeon, KOREA, REPUBLIC OF
Jang, Jong Soo, Daejeon, KOREA, REPUBLIC OF
Sohn, Sung Won, Daejeon, KOREA, REPUBLIC OF
Bang, Hyochan, Daejeon, KOREA, REPUBLIC OF

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003135759	A1	20030717	
APPLICATION INFO.:	US 2002-234207	A1	20020905	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	KR 2002-2465	20020116

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JACOBSON HOLMAN PLLC, 400 SEVENTH STREET N.W., SUITE 600, WASHINGTON, DC, 20004
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Page(s)
LINE COUNT: 794

L3 ANSWER 64 OF 104 USPATFULL on STN
TI Method and apparatus for dynamic client-side load balancing system
AB A method and apparatus for balancing load among a plurality of server computers connected via a network such as the Internet to a client computer. In one embodiment the invention includes a method of a client computer receiving plurality of addresses associated with a chosen Uniform Resource Locator. The method also includes identifying one of the plurality of addresses as a most recently used address and receiving a Uniform Resource Locator as an entered Uniform Resource Locator. The method further includes identifying the entered Uniform Resource Locator as a chosen Uniform Resource Locator and selecting from the plurality of addresses a selected address that is different from the most recently used address.

ACCESSION NUMBER: 2003:182417 USPATFULL
TITLE: Method and apparatus for dynamic client-side load balancing system
INVENTOR(S): Abir, Eli, South Salem, NY, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003126252	A1	20030703	
APPLICATION INFO.:	US 2002-233734	A1	20020904	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-316981P	20010905 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ARNOLD & PORTER, IP DOCKETING DEPARTMENT, RM 1126(b), 555 12TH STREET, N.W., WASHINGTON, DC, 20004-1206
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 692

L3 ANSWER 65 OF 104 USPATFULL on STN
TI IMPROVED MATERIALS AND METHODS FOR TRANSFORMATION
AB Disclosed herein are novel methods and materials directed to transforming a host cell and expressing exogenous RNA therein. Specifically disclosed are DNA-launching platforms used to introduce a replicating viral segment attached to an exogenous polynucleotide into a cell, whereby the exogenous polynucleotide is expressed in said cell and confers a detectable trait.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:107763 USPATFULL
TITLE: IMPROVED MATERIALS AND METHODS FOR TRANSFORMATION
INVENTOR(S): RASOCHOVA, LADA, MADISON, WI, UNITED STATES
GERMAN, THOMAS, HOLLANDALE, WI, UNITED STATES
AHLQUIST, PAUL, MADISON, WI, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003074677	A1	20030417
APPLICATION INFO.:	US 1999-316622	A1	19990521 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-86526P	19980522 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SALIWANCHIK LLOYD & SALIWANCHIK, A PROFESSIONAL ASSOCIATION, 2421 N.W. 41ST STREET, SUITE A-1, GAINESVILLE, FL, 326066669
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 2809
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 66 OF 104 USPATFULL on STN
TI Method and apparatus for estimating a geographic location of a networked entity
AB A method and an apparatus operates to associate a geographic location associated with a network address. At least one data collection operation is performed to obtain information pertaining to a network address. The retrieved information is processed to identify a plurality of geographic locations potentially associated with the network address, and to attach a confidence factor to each of the plurality of geographic locations. An estimated geographic location is selected from the plurality of geographic locations as being a best estimate of a true geographic location of the network address, where the selection of the estimated geographic location is based upon a degree of confidence-factor weighted agreement within the plurality of geographic locations.

ACCESSION NUMBER: 2003:107557 USPATFULL

TITLE: Method and apparatus for estimating a geographic location of a networked entity

INVENTOR(S): Anderson, Mark, Westminster, CO, UNITED STATES
Bansal, Ajay, Cupertino, CA, UNITED STATES
Doctor, Brad, Broomfield, CO, UNITED STATES
Hadjiyiannis, George, Cambridge, MA, UNITED STATES
Herringshaw, Christopher, West Wardsboro, VT, UNITED STATES
Karplus, Eli E., New Castle, CO, UNITED STATES
Muniz, Derald, Midlothian, TX, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003074471	A1	20030417	
	US 6684250	B2	20040127	
APPLICATION INFO.:	US 2001-825675	A1	20010403	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-194761P	20000403 (60)
	US 2000-241776P	20001018 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Andre I. Marais, BLAKELY, SOKOLOFF, TAYLOR & ZAFMAN LLP, Seventh Floor, 12400 Wilshire Boulevard, Los Angeles, CA, 90025-1026
NUMBER OF CLAIMS: 135
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 63 Drawing Page(s)
LINE COUNT: 3810

L3 ANSWER 67 OF 104 USPATFULL on STN
TI Network surveillance and security system
AB A system that monitors and protects the security of computer networks uses artificial intelligence, including learning algorithms, neural networks and genetic programming, to learn from security events. The invention maintains a knowledge base of security events that updates autonomously in real time. The invention encrypts communications to exchange changes in its knowledge base with separate security systems protecting other computer networks. The invention autonomously alters its security policies in response to ongoing events. The invention tracks network communication traffic from inception at a well-known port throughout the duration of the communication including monitoring of any port the communication is switched to. The invention is able to track and utilize UNIX processes for monitoring, threat detection, and threat response functions. The invention is able to subdivide the network communications into identifying tags for tracking and control of the communications without incurring lags in response times.

ACCESSION NUMBER: 2003:72739 USPATFULL
TITLE: Network surveillance and security system
INVENTOR(S): Carter, Ernst B., San Francisco, CA, UNITED STATES
Zolotov, Vasily, San Francisco, CA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003051026	A1	20030313	
APPLICATION INFO.:	US 2001-766560	A1	20010119	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: THOMPSON COBURN, LLP, ONE FIRSTAR PLAZA, SUITE 3500, ST LOUIS, MO, 63101
NUMBER OF CLAIMS: 40
EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 25 Drawing Page(s)
LINE COUNT: 5642

L3 ANSWER 68 OF 104 USPATFULL on STN
TI Method and apparatus for protecting electronic commerce from distributed denial-of-service attacks
AB An Internet Service Provider (ISP), in consideration of being remunerated in some manner by an e-merchant, carries the packets of a designated subset of that e-merchant's clients, designated as VIPs, in a privileged class of service as compared to an unprivileged class of service that is used to carry the packets of the e-merchant's other regular clients. In this way, the adverse effects on performance due to congestion in the unprivileged class of service, whether due to an ongoing denial-of-service attack or not, will not affect the performance of packets sent by and to VIPs using the privileged class of service. An e-merchant may select its VIPs from among those clients that bring in a majority of the e-merchant's revenues. An e-merchant turns a regular client into a VIP by granting it a VIP right. VIP gates, preferable implemented in an ISP's access gateways, monitor the packets sent by clients and mark for the privileged class of service those packets whose source has an active VIP right issued by the packet's destination.

ACCESSION NUMBER: 2003:52010 USPATFULL
TITLE: Method and apparatus for protecting electronic commerce from distributed denial-of-service attacks
INVENTOR(S): Brustoloni, Jose C., Westfield, NJ, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003036970	A1	20030220	
APPLICATION INFO.:	US 2002-175463	A1	20020619	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-313031P	20010816	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Docket Administrator (Room 3J-219), Lucent Technologies Inc., 101 Crawfords Corner Road, Holmdel, NJ, 07733-3030
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 801

L3 ANSWER 69 OF 104 USPATFULL on STN
TI Method and apparatus for protecting web sites from distributed denial-of-service attacks
AB An Internet Service Provider (ISP), in consideration of being remunerated in some manner by a site, determines whether packets destined to that site conform to a profile provided to the ISP by that site. The profile, indicates, for example, what protocols are allowed by the server, and, for each such protocol, what destination port numbers or message types are allowed, a maximum transmission rate, the maximum number of allowed connections a client may have, and whether to enforce congestion-avoidance. This server profile enforcement (SPE) automatically thwarts denial of service attacks from attackers that send packets to the subscribing server from that ISP using connections or having packet characteristics that do not conform to the acceptable characteristics specified in the profile. SPE is generally performed by an SPE unit, which can be incorporated in the access gateways of an ISP that supports the service. Packets may also be forwarded in multiple classes of service depending upon the type of traffic from which they originate. Multiple classes of service allow the method to be effective

even if deployed only by select ISPs.

ACCESSION NUMBER: 2003:50416 USPATFULL
TITLE: Method and apparatus for protecting web sites from distributed denial-of-service attacks
INVENTOR(S): Brustoloni, Jose?apos, C., Westfield, NJ, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003035370	A1	20030220	
APPLICATION INFO.:	US 2002-175458	A1	20020619	(10)'

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-313031P	20010816 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Lucent Technologies Inc., Docket Administrator (Room 3J-219), 101 Crawfords Corner Road, Holmdel, NJ, 07733-3030
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 906

L3 ANSWER 70 OF 104 USPATFULL on STN
TI Countermeasures for irregularities in financial transactions
AB A system and method for identifying financial transactions with the potential for financial irregularity (e.g. money laundering) comprises processing (20) financial transactions connected with a client, account and financial application, subjecting the client/account and transaction information to a set of rules (22) to produce numerical outcomes (116, 124, 132) indicative of the potential for money laundering being present. A user of the system is able to vary the weightings associated with each rule according to their importance to the particular circumstances of the institution in question.

ACCESSION NUMBER: 2003:45915 USPATFULL
TITLE: Countermeasures for irregularities in financial transactions
INVENTOR(S): Bosworth-Davies, Rowan, London, UNITED KINGDOM
Norfolk, Robert David, Worcester, UNITED KINGDOM
Burd, Paul, Tyler's Green, UNITED KINGDOM

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003033228	A1	20030213	
APPLICATION INFO.:	US 2001-998360	A1	20011129	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	GB 2000-29229	20001130

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: UNISYS Corporation, Unisys Way, MS/E8-114, Blue Bell, PA, 19424-0001
NUMBER OF CLAIMS: 65
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 1104

L3 ANSWER 71 OF 104 USPATFULL on STN
TI Method and apparatus for tracing packets in a communications network
AB A method for tracing packets in a communications network directed to

tracing a stream of anonymous packets received at a given target host, in order to identify their source, in response, for example, to a Denial-of-Service ("DoS") attack on the target host. Advantageously, the tracing is performed without reliance on knowledge or cooperation from intervening Internet Service Providers (ISPs) along the path. The method is performed by applying a "burst load" (i.e., a brief but heavy load of transmitted packets) to various elements (i.e., links or routers) in the network and measuring the change in the rate with which the stream of packets arrive at the target. If the rate is substantially altered upon introduction of the burst load, then it may be deduced that the given element is most likely on the path from the source host of the DoS attack to the target host.

ACCESSION NUMBER: 2003:11843 USPATFULL
TITLE: Method and apparatus for tracing packets in a communications network
INVENTOR(S): Burch, Hal Joseph, Pittsburgh, PA, UNITED STATES
Cheswick, William R., Bernardsville, NJ, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003009554	A1	20030109	
APPLICATION INFO.:	US 2001-901286	A1	20010709	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Lucent Technologies Inc., Docket Administrator (Room 3J-219), 101 Crawfords Corner Road, Holmdel, NJ, 07733
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 856

L3 ANSWER 72 OF 104 USPATFULL on STN
TI System and method for anti-network terrorism
AB Protecting a host network from a flood-type denial of service attack by passively collecting a data packet from data received by the host network, comparing information in the data packet to a signature of an attack type of the attack, and detecting the attack in response to a determination that the signature and the information comprise matching data. A defensive countermeasure can be initiated to protect the host network from the attack and to provide a pathway for an offensive countermeasure against a source of the attack.

ACCESSION NUMBER: 2002:296005 USPATFULL
TITLE: System and method for anti-network terrorism
INVENTOR(S): Lachman, John Paul, III, Singer Island, FL, UNITED STATES
Hsieh, Mansi, Los Angeles, CA, UNITED STATES
PATENT ASSIGNEE(S): Cyber Operations, LLC, Jupiter, FL, 33477 (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002166063	A1	20021107	
APPLICATION INFO.:	US 2002-86107	A1	20020228	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-272712P	20010301 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KING & SPALDING, 191 PEACHTREE STREET, N.E., ATLANTA, GA, 30303-1763
NUMBER OF CLAIMS: 70

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 41 Drawing Page(s)
LINE COUNT: 1992

L3 ANSWER 73 OF 104 USPATFULL on STN
TI METHOD AND APPARATUS FOR SOFTWARE TECHNICAL SUPPORT AND TRAINING
AB The present invention is a method and apparatus for supporting and training a user in operating a software application. A list of task indications are coupled to the GUI window. A graphical overlay is positioned on top of the GUI window and coupled to it. A sequence of instructions associated with a respective task is displayed in the graphical overlay upon selection of the task indication by the user. Each instruction directs attention to a respective selectable graphical area in the GUI window. The user operates a selector coupled to the GUI window, where after selecting a task, the selector is used to select graphical areas in response to the sequence of instructions. In the preferred embodiment, the present invention further comprises recorded voice files or a text-to-speech synthesizer coupled to the sequence of instructions, whereby the instruction being displayed is simultaneously presented audibly to the user. The selection of one or more selectable graphical areas in a sequence before selecting a task automatically highlights a list of possible tasks being performed. The present invention is capable of and well suited for operating a computer controlling a system such as a data communication network, where the tasks displayed in the list of task indications are user-privilege specific, and a password is used to restrict the list of task indications to a subset for display. The tasks displayed in the list of task indications are optionally presented to the user as a function of a mode setting, where a mode setting is a beginner, intermediate, or advanced mode setting. One advantage of a GUI coach over the prior art is that the user learns a sequence associated with a task through actively interfacing with the GUI window.

ACCESSION NUMBER: 2002:278732 USPATFULL
TITLE: METHOD AND APPARATUS FOR SOFTWARE TECHNICAL SUPPORT AND TRAINING
INVENTOR(S): MESSINGER, FREDERIC P., GROTON, MA, UNITED STATES
NEVIN, BRUCE E., EDGARTOWN, MA, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002154153	A1	20021024	
APPLICATION INFO.:	US 1999-345903	A1	19990701	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HAMILTON, BROOK, SMITH & REYNOLDS, P.C., 530 VIRGINIA ROAD, P.O. BOX 9133, CONCORD, MA, 01742-9133
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Page(s)
LINE COUNT: 867

L3 ANSWER 74 OF 104 USPATFULL on STN
TI Method and device for monitoring data traffic and preventing unauthorized access to a network
AB A method and device for protecting a network by monitoring both incoming and outgoing data traffic on multiple ports of the network, and preventing transmission of unauthorized data across the ports. The monitoring system is provided in an non-promiscuous mode and automatically denies access to data packets from a specific source based upon an associated rules table. All other packets from sources not violating the rules are allowed to use the same port. The monitoring system processes copies of the data packets resulting in minimal loss of throughput. The system is also highly adaptable and provides for dynamic

writing and issuing of firewall rules by updating the rules table. Information regarding the data packets is captured sorted and cataloged to determine attack profiles and unauthorized data packets.

ACCESSION NUMBER:	2002:244386 USPATFULL
TITLE:	Method and device for monitoring data traffic and preventing unauthorized access to a network
INVENTOR(S):	Shanklin, Carter, Woodland, CA, UNITED STATES Nadler, Michael, Sacramento, CA, UNITED STATES Ontiveros, Mark, Woodland, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002133586	A1	20020919
APPLICATION INFO.:	US 2001-844794	A1	20010427 (9)

RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2001-761499, filed on 16 Jan 2001, PENDING
DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	DKW LAW GROUP, P.C., 58TH FLOOR - USX TOWER, 600 GRANT STREET, PITTSBURGH, PA, 15219
NUMBER OF CLAIMS:	21
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	11 Drawing Page(s)
LINE COUNT:	1055

L3 ANSWER 75 OF 104 USPATFULL on STN

TI Scripted distributed denial-of-service (DDoS) attack discrimination using turing tests

AB A system, method and computer program product can include a test performed by a computer to determine whether a requestor of resources is a human user or a computer software scripted agent. If the test is passed, then the computer of the present invention assumes that the requestor of resources is a valid human user and access to resources is granted. In an exemplary embodiment of the present invention a system, method and computer program product for controlling access to resources. In an exemplary embodiment the method can include the steps of receiving a request from an entity; presenting the entity with a test; determining from the test whether or not the entity is an intelligent being; and granting the request only if the entity is determined to be an intelligent being.

ACCESSION NUMBER:	2002:222702 USPATFULL
TITLE:	Scripted distributed denial-of-service (DDoS) attack discrimination using turing tests
INVENTOR(S):	Tyree, David Spencer, Reston, VA, UNITED STATES
PATENT ASSIGNEE(S):	NETWORKS ASSOCIATES TECHNOLOGY, INC. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002120853	A1	20020829
APPLICATION INFO.:	US 2001-793733	A1	20010227 (9)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	Edward A. Pennington Esq., SWIDLER BERLIN SHEREFF FRIEDMAN, LLP, 3000 K Street, Suite 300, Washington, DC, 20007
NUMBER OF CLAIMS:	21
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	5 Drawing Page(s)
LINE COUNT:	969

L3 ANSWER 76 OF 104 USPATFULL on STN

TI Method and device for monitoring data traffic and preventing

unauthorized access to a network

AB A method and device for protecting a network by monitoring both incoming and outgoing data traffic on multiple ports of the network, and preventing transmission of unauthorized data across the ports. The monitoring system is provided in a non-promiscuous mode and automatically denies access to data packets from a specific source if it is determined that the source is sending unauthorized data (e.g., suspicious data or a denial of service attack). All other packets from sources not transmitting unauthorized data are allowed to use the same port. The monitoring system processes copies of the data packets resulting in minimal loss of throughput. The system is also highly adaptable and provides dynamic writing and issuing of firewall rules based on sample time and a threshold value for the number of packets transmitted. Information regarding the data packets is captured, sorted and cataloged to determine attack profiles and unauthorized data packets.

ACCESSION NUMBER:	2002:199839 USPATFULL			
TITLE:	Method and device for monitoring data traffic and preventing unauthorized access to a network			
INVENTOR(S):	Ontiveros, Mark, Woodland, CA, UNITED STATES Nadler, Michael H., Sacramento, CA, UNITED STATES			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002107953	A1	20020808	
APPLICATION INFO.:	US 2001-761499	A1	20010116	(9)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	DKW LAW GROUP, P.C., 58TH FLOOR - USX TOWER, 600 GRANT STREET, PITTSBURGH, PA, 15219			
NUMBER OF CLAIMS:	20			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	9 Drawing Page(s)			
LINE COUNT:	658			

L3 ANSWER 77 OF 104 USPATFULL on STN

TI Method and system for detecting unusual events and application thereof in computer intrusion detection

AB An automated decision engine is utilized to screen incoming alarms using a knowledge-base of decision rules. The decision rules are updated with the assistance of a data mining engine that analyzes historical data. "Normal" alarm events, sequences, or patterns generated by sensors under conditions not associated with unusual occurrences (such as intrusion attacks) are characterized and these characterizations are used to contrast normal conditions from abnormal conditions. By identifying frequent occurrences and characterizing them as "normal" it is possible to easily identify anomalies which would indicate a probable improper occurrence. This provides very accurate screening capability based on actual event data.

ACCESSION NUMBER:	2002:158272 USPATFULL			
TITLE:	Method and system for detecting unusual events and application thereof in computer intrusion detection			
INVENTOR(S):	Manganaris, Stefanos, Durham, NC, UNITED STATES Hermiz, Keith, Arlington, VA, UNITED STATES			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002082886	A1	20020627	
APPLICATION INFO.:	US 2000-749095	A1	20001227	(9)

	NUMBER	DATE

PRIORITY INFORMATION: US 2000-230486P 20000906 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Mark D. Simpson, Esquire, Synnestvedt & Lechner LLP, 2600 Aramark Tower, 1101 Market Street, Philadelphia, PA, 19107-2950
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 623

L3 ANSWER 78 OF 104 USPATFULL on STN
TI Lawn and guarding edging system (conduit extension)
AB The instant invention is a lawn and garden edging system which provides for a border device positionable adjacent walks and plant beds with provisions for the attachment of watering and lighting components. An insertion structure is provided which is inserted into the ground by use of a wooden handle placed into an aperture allowing an individual to stand on an upper end of the structure allowing the installer's weight to force the structure into the ground. The upper portion of the structure forms a passageway with apertures available for positioning watering components, electrical components may also be attached for connection to lighting products used to highlight trees, bushes or a home. A second embodiment of the invention provides a conduit for attachment to conventional extruded plastic edging dividers, the conduit provides the formation of a passageway for placement of the aforementioned watering components and electrical connections.

ACCESSION NUMBER: 2000:144451 USPATFULL
TITLE: Lawn and guarding edging system (conduit extension)
INVENTOR(S): Matz, Warren W., 13882 U.S. Hwy. 1, Juno Beach, FL, United States 33408

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6138405		20001031
APPLICATION INFO.:	US 1997-886373		19970701 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1996-680442, filed on 15 Jul 1996, now patented, Pat. No. US 5768824 which is a continuation-in-part of Ser. No. US 1995-435891, filed on 5 May 1995, now patented, Pat. No. US 5535545
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Carone, Michael J.
LEGAL REPRESENTATIVE: McHale & Slavin, P.A.
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 56 Drawing Figure(s); 32 Drawing Page(s)
LINE COUNT: 802

L3 ANSWER 79 OF 104 USPATFULL on STN
TI Lawn and garden edging system
AB The instant invention is a lawn and garden edging system which provides for a border device positionable adjacent walks and plant beds with provisions for the attachment of watering and lighting components. An insertion structure is provided which is inserted into the ground by use of a wooden handle placed into an aperture allowing an individual to stand on an upper end of the structure allowing the installer's weight to force the structure into the ground. The upper portion of the structure forms a passageway with apertures available for positioning watering components, electrical components may also be attached for connection to lighting products used to highlight trees, bushes or a home. A second embodiment of the invention provides a conduit for attachment to conventional extruded plastic edging dividers, the conduit

provides the formation of a passageway for placement of the aforementioned watering components and electrical connections.

ACCESSION NUMBER:	2000:14379 USPATFULL
TITLE:	Lawn and garden edging system
INVENTOR(S):	Matz, Warren W., 882 U.S. Hwy. 1, Juno Beach, FL, United States 33408

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6021599		20000208
APPLICATION INFO.:	US 1996-683646		19960715 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1995-435891, filed on 5 May 1995, now patented, Pat. No. US 5535545		

DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Carone, Michael J.
ASSISTANT EXAMINER:	Downs, Joanne C.
LEGAL REPRESENTATIVE:	McHale & Slavin
NUMBER OF CLAIMS:	8
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	44 Drawing Figure(s); 26 Drawing Page(s)
LINE COUNT:	719

L3 ANSWER 80 OF 104 USPATFULL on STN
TI Lawn and Garden edging system
AB The instant invention is a lawn and garden edging system which provides for a border device positionable adjacent walks and plant beds with provisions for the attachment of watering and lighting components. An insertion structure is provided which is inserted into the ground by use of a wooden handle placed into an aperture allowing an individual to stand on an upper end of the structure allowing the installer's weight to force the structure into the ground. The upper portion of the structure forms a passageway with apertures available for positioning watering components, electrical components may also be attached for connection to lighting products used to highlight trees, bushes or a home. A second embodiment of the invention provides a conduit for attachment to conventional extruded plastic edging dividers, the conduit provides the formation of a passageway for placement of the aforementioned watering components and electrical connections.

ACCESSION NUMBER:	1999:3177 USPATFULL
TITLE:	Lawn and Garden edging system
INVENTOR(S):	Matz, Warren W., 882 U.S. Highway 1, Juno Beach, FL, United States 33408

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5857493		19990112
APPLICATION INFO.:	US 1996-683647		19960715 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1995-435891, filed on 5 May 1995, now patented, Pat. No. US 5535545		

DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Brinson, Patrick F.
LEGAL REPRESENTATIVE:	McHale & Slavin PA
NUMBER OF CLAIMS:	10
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	44 Drawing Figure(s); 26 Drawing Page(s)
LINE COUNT:	718

L3 ANSWER 81 OF 104 USPATFULL on STN
TI Lawn and garden edging system
AB The instant invention is a lawn and garden edging system which provides

for a border device positionable adjacent walks and plant beds with provisions for the attachment of watering and lighting components. An insertion structure is provided which is inserted into the ground by use of a wooden handle placed into an aperture allowing an individual to stand on an upper end of the structure allowing the installer's weight to force the structure into the ground. The upper portion of the structure forms a passageway with apertures available for positioning watering components, electrical components may also be attached for connection to lighting products used to highlight trees, bushes or a home. A second embodiment of the invention provides a conduit for attachment to conventional extruded plastic edging dividers, the conduit provides the formation of a passageway for placement of the aforementioned watering components and electrical connections.

ACCESSION NUMBER:	1998:70907 USPATFULL
TITLE:	Lawn and garden edging system
INVENTOR(S):	Matz, Warren W., 882 U.S. Hwy. 1, Juno Beach, FL, United States 33408

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5768824		19980623
APPLICATION INFO.:	US 1996-680442		19960715 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1995-435891, filed on 5 May 1995, now patented, Pat. No. US 5535545		

DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Melius, Terry Lee
ASSISTANT EXAMINER:	Downs, Joanne C.
LEGAL REPRESENTATIVE:	McHale & Slavin
NUMBER OF CLAIMS:	11
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	45 Drawing Figure(s); 27 Drawing Page(s)
LINE COUNT:	741

L3 ANSWER 82 OF 104 USPATFULL on STN

TI Lawn and garden edging system

AB The instant invention is a lawn and garden edging system which provides for a border device positionable adjacent walks and plant beds with provisions for the attachment of watering and lighting components. An insertion structure is provided which is inserted into the ground by use of a wooden handle placed into an aperture allowing an individual to stand on an upper end of the structure allowing the installer's weight to force the structure into the ground. The upper portion of the structure forms a passageway with apertures available for positioning watering components, electrical components may also be attached for connection to lighting products used to highlight trees, bushes or a home. A second embodiment of the invention provides a conduit for attachment to conventional extruded plastic edging dividers, the conduit provides the formation of a passageway for placement of the aforementioned watering components and electrical connections.

ACCESSION NUMBER:	96:61948 USPATFULL
TITLE:	Lawn and garden edging system
INVENTOR(S):	Matz, Warren W., 882 U.S. Hwy. 1, Juno Beach, FL, United States 33408

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5535545		19960716
APPLICATION INFO.:	US 1995-435891		19950505 (8)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Melius, Terry Lee

ASSISTANT EXAMINER: Downs, Joanne C.
LEGAL REPRESENTATIVE: McHale & Slavin
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 44 Drawing Figure(s); 26 Drawing Page(s)
LINE COUNT: 726

L3 ANSWER 83 OF 104 BIOTECHDS COPYRIGHT 2004 THE THOMSON CORP. on STN
TI Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation; involving vector plasmid pCMV5-mediated gene transfer for expression in host cell
AN 2001-04474 BIOTECHDS
AB An isolated Smurf1 or Smurf2 protein (I), is claimed. Also claimed are: an isolated nucleic acid (II) encoding (I); a vector comprising (II); a host cell; production of (I); a transgenic non-human animal that expresses a human (I); screening for modulator of **Smurf** activity; an antibody that specifically binds to (I); an oligonucleotide or nucleic acid that specifically hybridizes to (II) under stringent conditions; and promoting a bone morphogenic protein or transforming growth factor (TGF)-beta activation pathway in a cell, comprising suppressing expression of endogenous **Smurf** in the cell. Expression of (I) from the vector in a cell is useful for inhibiting a bone morphogenic protein or TGF-beta activation pathway in a cell. (I) is useful to block chondrogenesis, osteogenesis, blood differentiation, cartilage formation, etc. (I) is useful for screening for various drugs and/or antibodies that can either enhance the bone morphogenic protein pathway, or inhibit it by antagonizing or mimicking the activity of (I), respectively. (I) is useful for treating a disorder associated with bone morphogenic protein or TGF-beta activation, such as cancer. (106pp)
ACCESSION NUMBER: 2001-04474 BIOTECHDS
TITLE: Novel isolated **Smurf** protein useful for inhibiting bone morphogenic protein or tumor growth factor-beta activation pathway, for treating cancer and to block osteogenesis, hair growth, tooth formation; involving vector plasmid pCMV5-mediated gene transfer for expression in host cell
AUTHOR: Thomsen G H; Wrana J
PATENT ASSIGNEE: Univ.New-York-State-Res.Found.; HSC-Res.Develop.
LOCATION: Toronto, Ontario, Canada.
PATENT INFO: WO 2000077168 21 Dec 2000
APPLICATION INFO: WO 2000-US16250 12 Jun 2000
PRIORITY INFO: US 1999-138969 11 Jun 1999
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2001-071267 [08]

L3 ANSWER 84 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Transforming Growth Factor-β Stimulates p300-dependent RUNX3 Acetylation, Which Inhibits Ubiquitination-mediated Degradation
AB The Runt domain transcription factors (RUNXs) play essential roles in normal development and neoplasias. Genetic analyses of animals and humans have revealed the involvement of RUNX1 in hematopoiesis and leukemia, RUNX2 in osteogenesis and cleidocranial dysplasia, and RUNX3 in the development of T-cells and dorsal root ganglion neurons and in the genesis of gastric cancer. Here we report that RUNX3 is a target of the acetyltransferase activity of p300. The p300-dependent acetylation of three lysine residues protects RUNX3 from ubiquitin ligase **Smurf** -mediated degradation The extent of the acetylation is up-regulated by the transforming growth factor-β signaling pathway and down-regulated by histone deacetylase activities. Our findings demonstrate that the level of RUNX3 protein is controlled by the competitive acetylation and deacetylation of the three lysine residues, revealing a new mechanism for

the posttranslational regulation of RUNX3 expression.

ACCESSION NUMBER: 2004:544572 HCAPLUS
DOCUMENT NUMBER: 141:86726
TITLE: Transforming Growth Factor-β Stimulates p300-dependent RUNX3 Acetylation, Which Inhibits Ubiquitination-mediated Degradation
AUTHOR(S): Jin, Yun-Hye; Jeon, Eun-Joo; Li, Qing-Lin; Lee, Yong Hee; Choi, Joong-Kook; Kim, Wun-Jae; Lee, Kwang-Youl; Bae, Suk-Chul
CORPORATE SOURCE: Departments of Biochemistry, School of Medicine and Institute for Tumor Research, Chungbuk National University, Cheongju, 361-763, S. Korea
SOURCE: Journal of Biological Chemistry (2004), 279(28), 29409-29417
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 85 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Pharmaceutical composition of interferon gamma with molecular diagnostics for the improved treatment of bronchial asthma
AB The disclosed invention relates to a novel pharmaceutical composition comprising interferon-γ and a diagnostic array of candidate polynucleotides for the improved treatment of lung diseases, especially for all forms of bronchial asthma. This invention describes the combination of mol. diagnosis and clin. therapy as a novel medication principle for reduction of mortality and improvement of disease management in bronchial asthma.

ACCESSION NUMBER: 2004:510134 HCAPLUS
DOCUMENT NUMBER: 141:52871
TITLE: Pharmaceutical composition of interferon gamma with molecular diagnostics for the improved treatment of bronchial asthma
INVENTOR(S): Bevec, Dorian; Ziesche, Rolf
PATENT ASSIGNEE(S): Mondobiotech Laboratories Anstalt, Liechtenstein
SOURCE: Eur. Pat. Appl., 23 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1430902	A1	20040623	EP 2002-28574	20021220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
PRIORITY APPLN. INFO.:			EP 2002-28574	20021220

L3 ANSWER 86 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Germline stem cell number in the Drosophila ovary is regulated by redundant mechanisms that control Dpp signaling
AB The available exptl. data support the hypothesis that the cap cells (CpCs) at the anterior tip of the germarium form an environmental niche for germline stem cells (GSCs) of the Drosophila ovary. Each GSC undergoes an asym. self-renewal division that gives rise to both a GSC, which remains associated with the CpCs, and a more posterior located cystoblast (CB). The CB upregulates expression of the novel gene, bag of marbles (bam), which is necessary for germline differentiation. Decapentaplegic (Dpp), a BMP2/4 homolog, has been postulated to act as a highly localized niche signal that maintains a GSC fate solely by repressing bam transcription.

Here, the authors further examine the role of Dpp in GSC maintenance. In contrast to the above model, the authors find that an enhancer trap inserted near the Dpp target gene, Daughters against Dpp (Dad), is expressed in addnl. somatic cells within the germarium, suggesting that Dpp protein may be distributed throughout the anterior germarium. However, Dad-lacZ expression within the germline is present only in GSCs and to a lower level in CBs, suggesting there are mechanisms that actively restrict Dpp signaling in germ cells. The authors demonstrate that one function of Bam is to block Dpp signaling downstream of Dpp receptor activation, thus establishing the existence of a neg. feedback loop between the action of the two genes. Moreover, in females doubly mutant for bam and the ubiquitin protein ligase **Smurf**, the number of germ cells responsive to Dpp is greatly increased relative to the number observed in either single mutant. These data indicate that there are multiple, genetically redundant mechanisms that act within the germline to downregulate Dpp signaling in the CB and its descendants, and raise the possibility that a CB and its descendants must become refractory to Dpp signaling in order for germline differentiation to occur.

ACCESSION NUMBER: 2004:455071 HCAPLUS
DOCUMENT NUMBER: 141:188183
TITLE: Germline stem cell number in the Drosophila ovary is regulated by redundant mechanisms that control Dpp signaling
AUTHOR(S): Casanueva, M. Olivia; Ferguson, Edwin L.
CORPORATE SOURCE: Committee on Developmental Biology, University of Chicago, Chicago, IL, 60637, USA
SOURCE: Development (Cambridge, United Kingdom) (2004), 131(9), 1881-1890
CODEN: DEVPED; ISSN: 0950-1991
PUBLISHER: Company of Biologists Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 87 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Ubiquitination on TGF-β signal transduction
AB A review, on the title study, discussing TGF-β/BMP signal transduction; Smurfs suppression of TGF-β/BMP signal transduction, Arkadia stimulation of TGF-β/BMP signaling; E3 ubiquitin ligase and others in regulation TGF-β/BMP signaling; and mechanism of Smad degradation and neoplasm.

ACCESSION NUMBER: 2004:400184 HCAPLUS
DOCUMENT NUMBER: 141:117257
TITLE: Ubiquitination on TGF-β signal transduction
AUTHOR(S): Imamura, Takeshi; Tajima, Yoshitaka; Koinuma, Daizo
CORPORATE SOURCE: Dep. of Biochemistry, Cancer Institute, Japan
SOURCE: Gendai Iryo (2004), 36(4), 837-843
CODEN: GEIRDK; ISSN: 0533-7259
PUBLISHER: Gendai Iryosha
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese

L3 ANSWER 88 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Impaired Smad7-**Smurf**-mediated negative regulation of TGF-β signaling in scleroderma fibroblasts
AB The principal effect of TGF-β1 on mesenchymal cells is its stimulation of ECM synthesis. Previous reports indicated the significance of the autocrine TGF-β loop in the pathogenesis of scleroderma. In this study, the authors focused on Smad7 and Smurfs, principal mols. in the neg. regulation of TGF-β signaling, to further understand the autocrine TGF-β loop in scleroderma. Scleroderma fibroblasts exhibited increased Smad7 levels compared with normal fibroblasts in vivo and in vitro. Smad7 constitutively formed a complex with the TGF-β

receptors, and the inhibitory effect of Smad7 on the promoter activity of human α2(I) collagen and 3TP-lux was completely impaired in scleroderma fibroblasts. Furthermore, the protein stability of TGF-β receptor type I was significantly increased in scleroderma fibroblasts compared with normal fibroblasts. There was no significant difference in Smurf1 and Smurf2 levels between normal and scleroderma fibroblasts, and the transiently overexpressed Smurf1 and/or Smurf2 did not affect TGF-β receptor type I protein levels in scleroderma fibroblasts. These results indicate that the impaired Smad7-**Smurf**-mediated inhibitory effect on TGF-β signaling might contribute to maintaining the autocrine TGF-β loop in scleroderma fibroblasts. To our knowledge, this is the first report of a disturbed neg. regulation of TGF-β signaling in fibrotic disorders.

ACCESSION NUMBER: 2004:64890 HCAPLUS
DOCUMENT NUMBER: 140:216014
TITLE: Impaired Smad7-**Smurf**-mediated negative regulation of TGF-β signaling in scleroderma fibroblasts
AUTHOR(S): Asano, Yoshihide; Ihn, Hironobu; Yamane, Kenichi; Kubo, Masahide; Tamaki, Kunihiko
CORPORATE SOURCE: Department of Dermatology, Faculty of Medicine, University of Tokyo, Tokyo, Japan
SOURCE: Journal of Clinical Investigation (2004), 113(2), 253-264
CODEN: JCINAO; ISSN: 0021-9738
PUBLISHER: American Society for Clinical Investigation
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 89 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Use of human RING finger protein 11 (RNF11 or PARK8) gene for diagnosis and treatment of late-onset idiopathic Parkinson's disease
AB The present invention provides human RING finger protein 11 gene (PARK8 gene or protein) for diagnosis and treatment of late-onset idiopathic Parkinson's disease. Polymorphisms associated in RNF 11 gene associated with increased susceptibility for Parkinson's disease are provided. Assays for screening for agents that alter the activity of a Parkinson's disease polypeptide (PARK8 or RNF11) or which identify PARK8 binding agents for therapy of Parkinson' s disease are disclosed.

ACCESSION NUMBER: 2003:737929 HCAPLUS
DOCUMENT NUMBER: 139:256363
TITLE: Use of human RING finger protein 11 (RNF11 or PARK8) gene for diagnosis and treatment of late-onset idiopathic Parkinson's disease
INVENTOR(S): Hicks, Andrew A.
PATENT ASSIGNEE(S): Decode Genetics Ehf., Iceland
SOURCE: PCT Int. Appl., 99 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003076658	A2	20030918	WO 2002-IB4276	20021014
WO 2003076658	A3	20031231		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,

UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: US 2002-363220P P 20020308

L3 ANSWER 90 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads
AB Smad ubiquitin regulatory factor (**Smurf**) 1 binds to receptor-regulated Smads for bone morphogenetic proteins (BMPs) Smad1/5 and promotes their degradation In addition, Smurf1 assocs. with transforming growth factor-β type I receptor through the inhibitory Smad (I-Smad) Smad7 and induces their degradation Herein, we examined whether Smurf1 neg. regulates BMP signaling together with the I-Smads Smad6/7. Smurf1 and Smad6 cooperatively induced secondary axes in Xenopus embryos. Using a BMP-responsive promoter-reporter construct in mammalian cells, we found that Smurf1 cooperated with I-Smad in inhibiting BMP signaling and that the inhibitory activity of Smurf1 was not necessarily correlated with its ability to bind to Smad1/5 directly. Smurf1 bound to BMP type I receptors via I-Smads and induced ubiquitination and degradation of these receptors. Moreover, Smurf1 associated with Smad1/5 indirectly through I-Smads and induced their ubiquitination and degradation Smurf1 thus controls BMP signaling with and without I-Smads through multiple mechanisms.
ACCESSION NUMBER: 2003:571354 HCAPLUS
DOCUMENT NUMBER: 139:302479
TITLE: Cooperative inhibition of bone morphogenetic protein signaling by Smurf1 and inhibitory Smads
AUTHOR(S): Murakami, Gyo; Watabe, Tetsuro; Takaoka, Kunio; Miyazono, Kohei; Imamura, Takeshi
CORPORATE SOURCE: Department of Biochemistry, The Cancer Institute of the Japanese Foundation for Cancer Research, Tokyo, 170-8455, Japan
SOURCE: Molecular Biology of the Cell (2003), 14(7), 2809-2817 CODEN: MBCEEV; ISSN: 1059-1524
PUBLISHER: American Society for Cell Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 91 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI dSmurf selectively degrades decapentaplegic-activated MAD, and its overexpression disrupts imaginal disc development
AB MAD plays an important role in decapentaplegic (DPP) signaling throughout Drosophila development. Despite a recent study describing the restriction of DPP signaling via putative ubiquitin E3 ligase dSmurf, the mol. mechanisms of how dSmurf affects DPP signaling remain unexplored. Toward this goal we demonstrated the degradation of phosphorylated MAD by dSmurf. DSmurf selectively interacted with MAD, but not Medea and Dad, and the MAD-dSmurf interaction was induced by constitutively active DPP type I receptor thickveins. Wild type dSmurf, but not its C1029A mutant, mediated ubiquitination-dependent degradation of MAD. Silencing of dSmurf using RNA interference stabilized MAD protein in Drosophila S2 cells. Targeted expression of dSmurf in various tissues abolished phosphorylated MAD and disrupted patterning and growth. In contrast, similar overexpression of inactive dSmurf(C1029A) showed no significant effects on development. We conclude that dSmurf specifically targets phosphorylated MAD to proteasome-dependent degradation and regulates DPP signaling during development.
ACCESSION NUMBER: 2003:536884 HCAPLUS
DOCUMENT NUMBER: 139:211039

TITLE: dSmurf selectively degrades decapentaplegic-activated MAD, and its overexpression disrupts imaginal disc development
AUTHOR(S): Liang, Yao-Yun; Lin, Xia; Liang, Min; Brunicardi, F. Charles; ten Dijke, Peter; Chen, Zhihong; Choi, Kwang-Wook; Feng, Xin-Hua
CORPORATE SOURCE: Department of Molecular & Cellular Biology, Michael E. DeBakey Department of Surgery, The Netherlands Cancer Institute, Amsterdam, 1066 CX, Neth.
SOURCE: Journal of Biological Chemistry (2003), 278(29), 26307-26310
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 92 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Cell cycle regulatory E3 ubiquitin ligases as anticancer targets
AB A review. Disregulation of the cell cycle and proliferation play key roles in cellular transformation and tumorigenesis. Such processes are intimately tied to the concentration, localization and activity of enzymes, adapters, receptors, and structural proteins in cells. Ubiquitination of these cellular regulatory proteins, governed by specific enzymes in the ubiquitin (Ub) conjugation cascade, has profound effects on their various functions, most commonly through proteasome targeting and degradation This review will focus on a variety of E3 Ub ligases as potential oncol. drug targets, with particular emphasis on the role of these mols. in the regulation of stability, localization, and activity of key proteins such as tumor suppressors and oncoproteins. E3 ubiquitin ligases that have established roles in cell cycle and apoptosis, such as the anaphase-promoting complex (APC), the Skp-1-Cull-F-box class, and the murine double minute 2 (MDM2) protein, in addition to more recently discovered E3 ubiquitin ligases which may be similarly important in tumorigenesis, (e.g. **Smurf** family, CHFR, and Efp), will be discussed. We will present evidence to support E3 ligases as good biol. targets in the development of anticancer therapeutics and address challenges in drug discovery for these targets.
ACCESSION NUMBER: 2003:130277 HCAPLUS
DOCUMENT NUMBER: 139:223432
TITLE: Cell cycle regulatory E3 ubiquitin ligases as anticancer targets
AUTHOR(S): Pray, Todd R.; Parlati, Francesco; Huang, Jianing; Wong, Brian R.; Payan, Donald G.; Bennett, Mark K.; Issakani, Sarkiz Daniel; Molineaux, Susan; Demo, Susan D.
CORPORATE SOURCE: Rigel Pharmaceuticals, Inc., South San Francisco, CA, 94080, USA
SOURCE: Drug Resistance Updates (2003), Volume Date 2002, 5(6), 249-258
CODEN: DRUPFW; ISSN: 1368-7646
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
REFERENCE COUNT: 81 THERE ARE 81 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 93 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI **Smurf** ubiquitin ligase in BMP signaling and development
AB Unavailable
ACCESSION NUMBER: 2002:920732 HCAPLUS
DOCUMENT NUMBER: 138:182929

TITLE: **Smurf** ubiquitin ligase in BMP signaling and development
AUTHOR(S): Zhu, Haitao
CORPORATE SOURCE: State Univ. of New York, Stony Brook, NY, USA
SOURCE: (2001) 141 pp. Avail.: UMI, Order No. DA3044980 From: Diss. Abstr. Int., B 2002, 63(3), 1190
DOCUMENT TYPE: Dissertation
LANGUAGE: English

L3 ANSWER 94 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Smad2 mediates transforming growth factor-β induction of endothelial nitric oxide synthase expression
AB Transforming growth factor-β (TGF-β) increases expression of endothelial nitric oxide synthase (eNOS), although the precise mechanism by which it does so is unclear. The authors report that Smad2, a transcription factor activated by TGF-β, mediates TGF-β induction of eNOS in endothelial cells. TGF-β induces Smad2 translocation from cytoplasm to nucleus, where it directly interacts with a specific region of the eNOS promoter. Overexpression of Smad2 increases basal levels of eNOS, and further increases TGF-β stimulation of eNOS expression. Ectopic expression of **Smurf**, an antagonizer of Smad2, decreases Smad2 expression and blocks TGF-β induction of eNOS. Because Smad2 can interact with a variety of transcription factors, coactivators, and corepressors, Smad2 may thus act as an integrator of multiple signals in the regulation of eNOS expression.
ACCESSION NUMBER: 2002:816005 HCAPLUS
DOCUMENT NUMBER: 138:131535
TITLE: Smad2 mediates transforming growth factor-β induction of endothelial nitric oxide synthase expression
AUTHOR(S): Saura, Marta; Zaragoza, Carlos; Cao, Wangsen; Bao, Clare; Rodriguez-Puyol, Manuel; Rodriguez-Puyol, Diego; Lowenstein, Charles J.
CORPORATE SOURCE: Department of Physiology, Universidad de Alcala, Madrid, Spain
SOURCE: Circulation Research (2002), 91(9), 806-813 CODEN: CIRUAL; ISSN: 0009-7330
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 95 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI A new partner for inhibitory Smads
AB A review discusses the mechanisms responsible for regulating the nuclear export of inhibitory Smads (I-Smads). A candidate for conducting nucleocytoplasmic shuttling of I-Smads is the **Smurf** family of ubiquitin E3 ligases, including Smurf1 and 2 in mammals. Smurf1 and 2 are HECT type E3 ubiquitin ligases, containing the N-terminal C2 domain, followed by WW domains and the C-terminal HECT domain. The interaction of Smurfs with I-Smads leads to the nuclear export of the latter. The nuclear export and membrane localization of I-Smads by Smurfs do not require the ubiquitin ligase activities of Smurfs, but occur independently of protein ubiquitination. Thus, Smurfs are most important partners for I-Smads permitting the latter to interact with serine/threonine kinase receptors.
ACCESSION NUMBER: 2001:922280 HCAPLUS
DOCUMENT NUMBER: 136:289097
TITLE: A new partner for inhibitory Smads
AUTHOR(S): Miyazono, Kohei
CORPORATE SOURCE: Department of Molecular Pathology, Graduate School of Medicine, University of Tokyo, Bunkyo-ku, Tokyo, 113-0033, Japan
SOURCE: Cytokine & Growth Factor Reviews (2002), 13(1), 7-9

CODEN: CGFRFB; ISSN: 1359-6101
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 96 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI The DSmurf ubiquitin-protein ligase restricts BMP signaling spatially and temporally during Drosophila embryogenesis
AB We identified Drosophila **Smurf** (DSmurf) as a neg. regulator of signaling by the BMP2/4 ortholog DPP during embryonic dorsal-ventral patterning. DSmurf encodes a HECT domain ubiquitin-protein ligase, homologous to vertebrate Smurf1 and Smurf2, that binds the Smad1/5 ortholog MAD and likely promotes its proteolysis. The essential function of DSmurf is restricted to its action on the DPP pathway. DSmurf has 2 distinct, possibly mechanistically sep., functions in controlling DPP signaling. Prior to gastrulation, DSmurf mutations cause a spatial increase in the DPP gradient, as evidenced by ventrolateral expansion in expression domains of target genes representing all known signaling thresholds. After gastrulation, DSmurf mutations cause a temporal delay in downregulation of earlier DPP signals, resulting in a lethal defect in hindgut organogenesis.
ACCESSION NUMBER: 2001:800471 HCAPLUS
DOCUMENT NUMBER: 136:82918
TITLE: The DSmurf ubiquitin-protein ligase restricts BMP signaling spatially and temporally during Drosophila embryogenesis
AUTHOR(S): Podos, Steven D.; Hanson, Kirsten K.; Wang, Yu-Chiun; Ferguson, Edwin L.
CORPORATE SOURCE: Department of Molecular Genetics and Cell Biology, University of Chicago, Chicago, IL, 60637, USA
SOURCE: Developmental Cell (2001), 1(4), 567-578
CODEN: DCEEBE; ISSN: 1534-5807
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 97 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI A new **Smurf** in the village
AB A review. TGF-β signaling is modulated by Smurfs, E3-ubiquitin ligases that selectively target the receptors and Smad proteins for degradation New evidence from Drosophila suggests that Smurfs regulate the amplitude and the duration of the cellular response to signaling in vivo.
ACCESSION NUMBER: 2001:800457 HCAPLUS
DOCUMENT NUMBER: 136:34940
TITLE: A new **Smurf** in the village
AUTHOR(S): Arora, K.; Warrior, R.
CORPORATE SOURCE: Department of Developmental and Cell Biology, University of California, Irvine, Irvine, CA, 92697, USA
SOURCE: Developmental Cell (2001), 1(4), 441-442
CODEN: DCEEBE; ISSN: 1534-5807
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

L3 ANSWER 98 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI sequences human ubiquitin-protein synthetases as antagonists of BMP and TGF<b signaling pathways and expression during development and interactions with Smad proteins
AB This invention provides unique members of the Hect family of ubiquitin

ligases that specifically target BMP and TGF<b/activin pathway-specific Smads. The novel ligases have been named Smurf1 and Smurf2. A transgenic expression system is described for these two proteins. They directly interact with Smads1 and 5 and Smad7, resp., and regulate the ubiquitination, turnover and activity of Smads and other proteins of these pathways. Smurf1 interferes with biol. responses to BMP, but not activin signaling. In amphibian embryos Smurf1 inhibits endogenous BMP signals, resulting in altered pattern formation and cell fate specification in the mesoderm and ectoderm. The present invention provides a unique regulatory link between the ubiquitination pathway and the control of cell fate determination
by the TGF<b superfamily during embryonic development. Thus, Smurf1 is a neg. regulator of Smad1 signal transduction, by targeting Smad1, Smurf1 blocks BMP signaling. Screening assays which survey **Smurf** WW domain interaction with Smad protein PPXY domain are also relayed. In mammalian cells, Smurf2 suppresses TGF<b signaling, and in Xenopus, blocks formation of dorsal mesoderm and causes anterior truncation of the embryos. Smurf2 forms a stable complex with Smad7, which induces degradation and downregulation of TGF<b/activin signaling. The human Smurf1 gene was mapped to 7q21.1-q31.1.

ACCESSION NUMBER: 2000:900772 HCAPLUS
DOCUMENT NUMBER: 134:53133
TITLE: sequences human ubiquitin-protein synthetases as antagonists of BMP and TGF<b signaling pathways and expression during development and interactions with Smad proteins
INVENTOR(S): Thomsen, Gerald H.; Wrana, Jeffrey
PATENT ASSIGNEE(S): Research Foundation of State University of New York, USA; HSC Research and Development Limited Partnership
SOURCE: PCT Int. Appl., 106 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000077168	A2	20001221	WO 2000-US16250	20000612
WO 2000077168	A3	20010503		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2000056107	A5	20010102	AU 2000-56107	20000612
EP 1192174	A2	20020403	EP 2000-941398	20000612
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: US 1999-138969P P 19990611
WO 2000-US16250 W 20000612

L3 ANSWER 99 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Sweetness and salivary sweetener concentration: a time-intensity study
AB A time- intensity study of sweetness of glucose syrup solns. was conducted by the "**SMURF**" technique. Both intensity and persistence of sweetness declined with increasing chain length (DP) of glucose syrup solution but persistence lasted up to 79 s whereas all carbohydrates had declined to sub-recognition threshold concns. in saliva by 20 s. This behavior is in sharp contrast to that of saccharin. Apparent sp. volume of

oligosaccharides decline with increasing DP, indicating better hydration. This leads to better receptor recruitment and thus greater intensity and persistence of sweetness in longer chain mols.

ACCESSION NUMBER: 2000:609950 HCAPLUS
DOCUMENT NUMBER: 133:295603
TITLE: Sweetness and salivary sweetener concentration: a time-intensity study
AUTHOR(S): Birch, G. G.; Karim, R.; Raymond, T.
CORPORATE SOURCE: The Department of Food Science and Technology, The University of Reading, Reading, RG6 6AP, UK
SOURCE: ACS Symposium Series (2000), 763(Flavor Release), 395-404
CODEN: ACSMC8; ISSN: 0097-6156
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 100 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Sweetness and salivary sweetner concentration: A time-intensity study.
AB A time-intensity study of the sweetness of glucose syrup solns. was conducted by the "**SMURF**" technique.Both intensity and persistence of sweetness declined with increasing chain length (DP) of glucose syrup solution but persistence lasted up to 79s whereas all carbohydrates had declined to sub-recognition threshold concns. in saliva by 20s.This behavior is in sharp contrast to that of saccharin.Apparent sp. vol.s of oligosaccharides decline with increasing DP, indicating better hydration.This leads to better receptor recruitment and thus greater intensity and persistence of sweetness in longer chain mols.

ACCESSION NUMBER: 1999:539331 HCAPLUS
TITLE: Sweetness and salivary sweetner concentration: A time-intensity study.
AUTHOR(S): Birch, Gordon G.; Karim, R.; Raymond, T.
CORPORATE SOURCE: Dept. of Food Science and Technology, University of Reading, Reading, Berks, RG6 6AP, UK
SOURCE: Book of Abstracts, 218th ACS National Meeting, New Orleans, Aug. 22-26 (1999), AGFD-096. American Chemical Society: Washington, D. C.
CODEN: 67ZJA5
DOCUMENT TYPE: Conference; Meeting Abstract
LANGUAGE: English

L3 ANSWER 101 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI The hydrostatic and hydrodynamic volumes of polyols in aqueous solutions and their sweet taste
AB The tastes and solution properties of sugar alcs. were studied in an attempt to illuminate the mechanism of sweet taste chemoreception. The **SMURF** method was used to measure taste time-intensity of aqueous solns. of sugar alcs. and the results were interpreted using the Stevens power function and kinetic parameters. The apparent molar volumes, apparent sp. vol.s, partial molar volumes, partial sp. vols. and intrinsic viscosities of the solns. were studied. Apparent molar volume reflects the size of the mol. in a hydrostatic state whereas intrinsic viscosity gives a measure of the size of the mols. in a hydrodynamic state. Generally the apparent molar volumes of the polyols are 6-13% greater than those of the parent sugars, indicating less interaction with the water structure. Apparent sp. volume values can predict taste quality, and the average apparent sp. volume for the sugar alcs. studied fits within the central part of the sweet range, i.e. 0.5-0.68 cm3/g, which accords with their ability to elicit a pure sweet taste response. Intensities and persistences of sweetness in the polyols followed the same trend as intrinsic viscosities.

ACCESSION NUMBER: 1997:325680 HCAPLUS

DOCUMENT NUMBER: 127:33061
TITLE: The hydrostatic and hydrodynamic volumes of polyols in aqueous solutions and their sweet taste
AUTHOR(S): Chavez, Atala Lopez; Birch, Gordon G.
CORPORATE SOURCE: Department of Agriculture and Food Technology, ITESM-Campus Queretaro, Queretaro, Mex.
SOURCE: Chemical Senses (1997), 22(2), 149-161
CODEN: CHSED8; ISSN: 0379-864X
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 102 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Solute-solvent interactions and the sweet taste of small carbohydrates. Part II: sweetness intensity and persistence in ethanol-water mixtures
AB Intensity and persistence of sweet taste of sugars (glucose, fructose, xylose and sucrose) and polyols (sorbitol, xylitol) were determined in ethanol-water mixts. using a sensory measuring unit for recording flux (**SMURF**) device. In all cases sweetness intensity and persistence were decreased when ethanol concentration was increased from 10% to 30%. Assessment of intensity/time responses for varied (2.3-9.2%, weight/volume) concns. of D-glucose, D-fructose and sucrose in 5% ethanol mixture showed that persistence is more affected by the presence of ethanol than intensity. These results are interpreted by the solution properties in the ethanol-water binary solvent.
ACCESSION NUMBER: 1993:37723 HCAPLUS
DOCUMENT NUMBER: 118:37723
TITLE: Solute-solvent interactions and the sweet taste of small carbohydrates. Part II: sweetness intensity and persistence in ethanol-water mixtures
AUTHOR(S): Hoopman, Tineke; Birch, Gordon; Serghat, Samira; Portmann, Marie Odile; Mathlouthi, Mohamed
CORPORATE SOURCE: Dep. Food Sci. Technol., Univ. Reading, Whiteknights/Reading, RG6 2AP, UK
SOURCE: Food Chemistry (1993), 46(2), 147-53
CODEN: FOCHDJ; ISSN: 0308-8146
DOCUMENT TYPE: Journal
LANGUAGE: English

L3 ANSWER 103 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Study of some factors affecting intensity/time characteristics of sweetness
AB Intensity/time plots of sweetness produced by D-glucose, D-fructose and sucrose at concns. ranging from 2.3 to 9.2% (weight/volume) were recorded for solns. at 15, 22 and 35°. The intensity (I) and persistence (P) power functions were applied to the results obtained with a potentiometer connected to a chart recorder similar to the sensory measurement unit recording flux (**SMURF**) device. Increasing the concentration of assessed samples leads to an increase of perceived intensity with a tendency to show a compression for D-fructose and sucrose and an expansion for D-glucose. Persistence increases linearly as a function of concentration for the three sugars. Only very slight modification of intensity and persistence are observed when the temperature is varied from 15 to 35°. Intensity/time plots were also recorded to 22° for solns. containing 5% sucrose or equisweet concns. of D-glucose or D-fructose brought to apparent viscosities of 5, 15, 25 and 35 mPa by addition of maltodextrins. The sweetness intensity decreases as viscosity increases for D-fructose and sucrose solns. whereas it remains constant for D-glucose. The persistence remains almost constant for the three sugars when the viscosity is varied. A decrease in intrinsic viscosity [η], B-coeffs. and hydration nos. is obsd with increasing temperature while the apparent specific

volume is increased. From the Raman spectra of water and aqueous solns. of sugars, it may be concluded that increasing the temperature leads to a lowering of the rigidity of the hydrogen bonded clusters and an increase in mobility of H2O mols. The increase in the size of the sugars derived from apparent specific volume values reduces their accessibility to the receptor site. This effect is minimized as regards the perceived sweetness by the increased mobility of water. The effects of concentration temperature and viscosity on the intensity and persistence of the sweet taste of D-glucose, D-fructose and sucrose, together with their physicochem. properties in dilute solution, suggest that the accessibility of the sweet mol. to the receptor is an important step in the taste chemoreception. This step is followed by a biochem. phenomenon involving opening of ion channels which is sensitive to the mobility of water and around the site and the sweetener.

ACCESSION NUMBER: 1992:211850 HCAPLUS
DOCUMENT NUMBER: 116:211850
TITLE: Study of some factors affecting intensity/time characteristics of sweetness
AUTHOR(S): Portmann, Marie Odile; Serghat, Samira; Mathlouthi, Mohamed
CORPORATE SOURCE: Fac. Sci., Univ. Reims-Champagne-Ardenne, Reims, 511062, Fr.
SOURCE: Food Chemistry (1992), 44(2), 83-92
CODEN: FOCHDJ; ISSN: 0308-8146
DOCUMENT TYPE: Journal
LANGUAGE: English

L3 ANSWER 104 OF 104 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Role of the anomeric center in sugar sweetness
AB Intensity-time studies, using the sensory measuring unit for recording flux (**SMURF**), of sweetness of D-glucose solns. showed that there were no major differences between α- and β-anomers. Nor did the α- and β-anomers exhibit differences in apparent molar volumes. Contrary to previous reports, the anomeric center of D-fructose may play no direct role in the sweetness response.

ACCESSION NUMBER: 1987:64981 HCAPLUS
DOCUMENT NUMBER: 106:64981
TITLE: Role of the anomeric center in sugar sweetness
AUTHOR(S): Birch, G. G.; Shamil, S.; Shepherd, Z.
CORPORATE SOURCE: Natl. Coll. Food Technol., Univ. Reading, Reading, RG6 2AP, UK
SOURCE: Experientia (1986), 42(11-12), 1232-4
CODEN: EXPEAM; ISSN: 0014-4754
DOCUMENT TYPE: Journal
LANGUAGE: English

=> s smad with smurf
L4 1 SMAD WITH SMURF

=> d l4 ti abs ibib tot

L4 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2004 ACS on STN
TI A new partner for inhibitory Smads
AB A review discusses the mechanisms responsible for regulating the nuclear export of inhibitory Smads (I-Smads). A candidate for conducting nucleocytoplasmic shuttling of I-Smads is the Smurf family of ubiquitin E3 ligases, including Smurf1 and 2 in mammals. Smurf1 and 2 are HECT type E3 ubiquitin ligases, containing the N-terminal C2 domain, followed by WW domains and the C-terminal HECT domain. The interaction of Smurfs with I-Smads leads to the nuclear export of the latter. The nuclear export and membrane localization of I-Smads by Smurfs do not require the ubiquitin ligase activities of Smurfs, but occur independently of protein ubiquitination. Thus, Smurfs are most important partners for I-Smads

permitting the latter to interact with serine/threonine kinase receptors.
ACCESSION NUMBER: 2001:922280 HCAPLUS
DOCUMENT NUMBER: 136:289097
TITLE: A new partner for inhibitory Smads
AUTHOR(S): Miyazono, Kohei
CORPORATE SOURCE: Department of Molecular Pathology, Graduate School of Medicine, University of Tokyo, Bunkyo-ku, Tokyo, 113-0033, Japan
SOURCE: Cytokine & Growth Factor Reviews (2002), 13(1), 7-9
CODEN: CGFRFB; ISSN: 1359-6101
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s Nedd4 and WW domain
L5 1872 NEDD4 AND WW DOMAIN

=> s l5 and PY motif
L6 61 L5 AND PY MOTIF

=> s l6 and smurf
L7 0 L6 AND SMURF

=> s l6 and ppyx domain
L8 0 L6 AND PPYX DOMAIN

=> s l6 and smad
L9 5 L6 AND SMAD

=> d l9 ti abs ibib tot

L9 ANSWER 1 OF 5 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Screening for modulators of TGF-beta and/or bone morphogenic protein (BMP) mediated signaling useful for treating cancer and osteoporosis by evaluating the ability of agents to modulate **Smad** protein degradation -
AN AAB83021 peptide DGENE
AB The present sequence is the **WW domain** of a HECT (homologous to E6 carboxyl terminus) E3 ubiquitin ligase. The **WW domain** binds to the **Smad PY motif**, resulting in ubiquitination of **Smad** by the E3 ubiquitin ligase. The sequence is provided in a specification relating to a method for screening for agents that modulate transforming growth factor (TGF)-beta and/or bone morphogenic protein (BMP)-mediated signalling. The method involves evaluating the effect of an agent on binding of HECT E3 ubiquitin ligase **WW domain** to **Smad PY motif**, on ubiquitination of **Smad** protein by E3 ubiquitin ligase, or on the cellular levels of **Smad** protein HECT E3 ubiquitin ligase activity. The method is useful for stimulating bone formation in a patient or treating a condition associated with insufficient TGF-beta and/or BMP-mediated cell signalling. Agents that inhibit BMP-mediated signalling are useful for treating inflammation, ageing, cancer and infectious diseases. Agents that augment BMP-mediated signalling are useful for stimulating bone anabolism as well as treating broken bones, osteoporosis, and acute or chronic renal failure. Agents that inhibit TGF-mediated signalling are useful for treating cancer, inflammation, neurodegeneration and fibrosis.
ACCESSION NUMBER: AAB83021 peptide DGENE
TITLE: Screening for modulators of TGF-beta and/or bone morphogenic protein (BMP) mediated signaling useful for treating cancer and osteoporosis by evaluating the ability of agents to

modulate **Smad** protein degradation -
INVENTOR: Hoekstra M F; Xie W; Murray B W; Mercurio F M
PATENT ASSIGNEE: (SIGN-N)SIGNAL PHARM INC.
PATENT INFO: WO 2001016604 A1 20010308 75p
APPLICATION INFO: WO 2000-US23729 20000829
PRIORITY INFO: US 1999-385918 19990830
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-327913 [34]
DESCRIPTION: Human **Nedd4** HECT E3 ubiquitin ligase **WW domain** #4.

L9 ANSWER 2 OF 5 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Screening for modulators of TGF-beta and/or bone morphogenic protein (BMP) mediated signaling useful for treating cancer and osteoporosis by evaluating the ability of agents to modulate **Smad** protein degradation -
AN AAB83020 peptide DGENE
AB The present sequence is the **WW domain** of a HECT (homologous to E6 carboxyl terminus) E3 ubiquitin ligase. The **WW domain** binds to the **Smad PY motif**, resulting in ubiquitination of **Smad** by the E3 ubiquitin ligase. The sequence is provided in a specification relating to a method for screening for agents that modulate transforming growth factor (TGF)-beta and/or bone morphogenic protein (BMP)-mediated signalling. The method involves evaluating the effect of an agent on binding of HECT E3 ubiquitin ligase **WW domain** to **Smad PY motif**, on ubiquitination of **Smad** protein by E3 ubiquitin ligase, or on the cellular levels of **Smad** protein HECT E3 ubiquitin ligase activity. The method is useful for stimulating bone formation in a patient or treating a condition associated with insufficient TGF-beta and/or BMP-mediated cell signalling. Agents that inhibit BMP-mediated signalling are useful for treating inflammation, ageing, cancer and infectious diseases. Agents that augment BMP-mediated signalling are useful for stimulating bone anabolism as well as treating broken bones, osteoporosis, and acute or chronic renal failure. Agents that inhibit TGF-mediated signalling are useful for treating cancer, inflammation, neurodegeneration and fibrosis.
ACCESSION NUMBER: AAB83020 peptide DGENE
TITLE: Screening for modulators of TGF-beta and/or bone morphogenic protein (BMP) mediated signaling useful for treating cancer and osteoporosis by evaluating the ability of agents to modulate **Smad** protein degradation -
INVENTOR: Hoekstra M F; Xie W; Murray B W; Mercurio F M
PATENT ASSIGNEE: (SIGN-N)SIGNAL PHARM INC.
PATENT INFO: WO 2001016604 A1 20010308 75p
APPLICATION INFO: WO 2000-US23729 20000829
PRIORITY INFO: US 1999-385918 19990830
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-327913 [34]
DESCRIPTION: Human **Nedd4** HECT E3 ubiquitin ligase **WW domain** #3.

L9 ANSWER 3 OF 5 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Screening for modulators of TGF-beta and/or bone morphogenic protein (BMP) mediated signaling useful for treating cancer and osteoporosis by evaluating the ability of agents to modulate **Smad** protein degradation -
AN AAB83019 peptide DGENE
AB The present sequence is the **WW domain** of a HECT (homologous to E6 carboxyl terminus) E3 ubiquitin ligase. The **WW domain** binds to the **Smad PY motif**, resulting in ubiquitination of **Smad** by the E3 ubiquitin ligase.

The sequence is provided in a specification relating to a method for screening for agents that modulate transforming growth factor (TGF)-beta and/or bone morphogenic protein (BMP)-mediated signalling. The method involves evaluating the effect of an agent on binding of HECT E3 ubiquitin ligase **WW domain** to **Smad** **PY motif**, on ubiquitination of **Smad** protein by E3 ubiquitin ligase, or on the cellular levels of **Smad** protein HECT E3 ubiquitin ligase activity. The method is useful for stimulating bone formation in a patient or treating a condition associated with insufficient TGF-beta and/or BMP-mediated cell signalling. Agents that inhibit BMP-mediated signalling are useful for treating inflammation, ageing, cancer and infectious diseases. Agents that augment BMP-mediated signalling are useful for stimulating bone anabolism as well as treating broken bones, osteoporosis, and acute or chronic renal failure. Agents that inhibit TGF-mediated signalling are useful for treating cancer, inflammation, neurodegeneration and fibrosis.

ACCESSION NUMBER: AAB83019 peptide DGENE
TITLE: Screening for modulators of TGF-beta and/or bone morphogenic protein (BMP) mediated signaling useful for treating cancer and osteoporosis by evaluating the ability of agents to modulate **Smad** protein degradation -
INVENTOR: Hoekstra M F; Xie W; Murray B W; Mercurio F M
PATENT ASSIGNEE: (SIGN-N)SIGNAL PHARM INC.
PATENT INFO: WO 2001016604 A1 20010308 75p
APPLICATION INFO: WO 2000-US23729 20000829
PRIORITY INFO: US 1999-385918 19990830
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-327913 [34]
DESCRIPTION: Human **Nedd4** HECT E3 ubiquitin ligase **WW domain** #2.

L9 ANSWER 4 OF 5 DGENE COPYRIGHT 2004 The Thomson Corp on STN
TI Screening for modulators of TGF-beta and/or bone morphogenic protein (BMP) mediated signaling useful for treating cancer and osteoporosis by evaluating the ability of agents to modulate **Smad** protein degradation -
AN AAB83018 peptide DGENE
AB The present sequence is the **WW domain** of a HECT (homologous to E6 carboxyl terminus) E3 ubiquitin ligase. The **WW domain** binds to the **Smad** **PY motif**, resulting in ubiquitination of **Smad** by the E3 ubiquitin ligase. The sequence is provided in a specification relating to a method for screening for agents that modulate transforming growth factor (TGF)-beta and/or bone morphogenic protein (BMP)-mediated signalling. The method involves evaluating the effect of an agent on binding of HECT E3 ubiquitin ligase **WW domain** to **Smad** **PY motif**, on ubiquitination of **Smad** protein by E3 ubiquitin ligase, or on the cellular levels of **Smad** protein HECT E3 ubiquitin ligase activity. The method is useful for stimulating bone formation in a patient or treating a condition associated with insufficient TGF-beta and/or BMP-mediated cell signalling. Agents that inhibit BMP-mediated signalling are useful for treating inflammation, ageing, cancer and infectious diseases. Agents that augment BMP-mediated signalling are useful for stimulating bone anabolism as well as treating broken bones, osteoporosis, and acute or chronic renal failure. Agents that inhibit TGF-mediated signalling are useful for treating cancer, inflammation, neurodegeneration and fibrosis.

ACCESSION NUMBER: AAB83018 peptide DGENE
TITLE: Screening for modulators of TGF-beta and/or bone morphogenic protein (BMP) mediated signaling useful for treating cancer and osteoporosis by evaluating the ability of agents to modulate **Smad** protein degradation -
INVENTOR: Hoekstra M F; Xie W; Murray B W; Mercurio F M

PATENT ASSIGNEE: (SIGN-N)SIGNAL PHARM INC.
PATENT INFO: WO 2001016604 A1 20010308 75p
APPLICATION INFO: WO 2000-US23729 20000829
PRIORITY INFO: US 1999-385918 19990830
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2001-327913 [34]
DESCRIPTION: Human **Nedd4** HECT E3 ubiquitin ligase **WW domain** #1.

L9 ANSWER 5 OF 5 USPATFULL on STN
TI Methods for modulating signal transduction mediated by TGF-beta related proteins
AB Methods are provided for identifying agents that modulate signaling mediated by transforming growth factor beta (TGF-β) and members of the TGF-β family, such as bone morphogenic protein (BMP). Such agents may be identified using screens that evaluate candidate agents for the ability to modulate **Smad** protein degradation. Agents identified as described herein may be used to augment or inhibit signaling mediated by one or more TGF-β family members in a variety of cell types and for therapeutic purposes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:173228 USPATFULL
TITLE: Methods for modulating signal transduction mediated by TGF-beta related proteins
INVENTOR(S): Hoekstra, Merl F., Cardiff-by-the-sea, CA, UNITED STATES
Xie, Weilin, San Diego, CA, UNITED STATES
Murray, Brion W., San Diego, CA, UNITED STATES
Mercurio, Frank M., Del Mar, CA, UNITED STATES
PATENT ASSIGNEE(S): Signal Pharmaceuticals, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003119072	A1	20030626
APPLICATION INFO.:	US 2002-307956	A1	20021202 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 1999-385918, filed on 30 Aug 1999, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PENNIE AND EDMONDS, 1155 AVENUE OF THE AMERICAS, NEW YORK, NY, 100362711
NUMBER OF CLAIMS: 54
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 1625
CAS INDEXING IS AVAILABLE FOR THIS PATENT.